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THE NORMAL ELECTROCARDIOGRAM

BY

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From the Heart Department, the Royal Infirmary, Liverpool

Received November 18, 1938

It is necessary at intervals to revise our standards of the normal in all sciences. This is particularly desirable in electrocardiography, where important advances are made yearly in its application to the diagnosis of cardiac disease. It is very easy for minor changes in the electrocardiogram to loom too largely in the diagnostic picture ; but if the wide limits of normal variation are realized, some of the pitfalls will be avoided.

The most important early work on the normal electrocardiogram is that of Lewis (1912), who constructed tables giving the amplitude of various waves and the length of the various intervals usually measured in the human electrocardiogram. His work was carried out on fifty healthy subjects and the description of the characteristics of each wave forms the basis of our present-day interpretation of the electrocardiogram.

Another and more recent important contribution was the study of the electrocardiogram in late middle life by Jensen, Smith, and Cartwright (1931–2). Their investigation consisted of the analysis of fifty cases between the ages of fifty and sixty-five to determine if there were " any changes which, though not common in the tracings of younger people, could nevertheless be considered physiologic at this stage of life". Their conclusions are summarized below.

(1) The following findings were not considered pathological :

(*a*) Diphasic or iso-electric P waves in lead I, or inverted P waves in lead III if they become upright on deep inspiration.

(*b*) Slurring of the QRS complexes, especially in lead III, and slight to moderate notching of R, with the QRS interval below 0·10 sec.

(*c*) " Transverse heart."

(*d*) Isolated left ventricular preponderance.

(*e*) Moderate inversion of T in lead III.

(2) The following findings, on the other hand, were not present in their series and were therefore to be looked upon with suspicion :

(*a*) Indeterminate or inverted P waves in leads I or II.

(*b*) Inverted P waves in lead III if they did not become upright on deep inspiration.

(*c*) P-R interval exceeding 0·20 of a second, QRS interval exceeding 0·10, or S-T exceeding 0·34–0·36 of a second (the upper limit seems slightly uncertain).

They conclude that there did not seem to be any characteristic changes in the electrocardiogram with increasing age. The writers of the present paper felt that a larger series of cases might indicate whether some of the minor defects sometimes taken as pathological could, in reality, be physiological. At the same time it was hoped to make a more extensive study of the variation of the electrocardiogram with age.

Clinical Material

Each subject was, as far as possible, subjected to such a clinical examination as to exclude the likelihood of any cardiovascular disease or of any disease likely to affect the cardiovascular system temporarily. A large number were nurses and medical students apparently in perfect health. Others were patients in hospital for trivial surgical causes, and in them a generous interval was allowed after any operative interference had been undertaken. All cases that had any symptoms suggestive of cardiac involvement, any rise of blood pressure, any enlargement of the heart, or any murmurs which were not clearly functional were rigorously excluded. Further, no one who had suffered from rheumatic fever, chorea, repeated tonsillitis, syphilis, or other ætiological factors of heart disease was included. The Wassermann reaction was performed in each and the subject excluded if it was positive.

In all, 302 subjects were examined of whom more than half were in the third and fourth decades, a period of life when the cardiovascular system is likely to be intact. Measurements were made of the amplitude of P, Q, R, S, and T in all three leads. The time intervals P-R, QRS, and S-T were measured only in lead II. Details of these figures are to be seen in the accompanying tables.

Results

The results may be summarized as regards the various waves, axis deviation, and the time intervals.

P Wave.—In a number of instances P 1 was flat or scarcely perceptible. P 2 was usually well developed, and P 3 variable. Inverted P waves of a well-defined character were only observed in lead III. Four examples were noted in the second, third, and fifth decades. Occasional splitting of the summit of the P wave was noticed, not unlike that found more frequently in mitral stenosis.

Fig. 1.—Types of P wave : (A) P with broad summit ; (B) Bifurcation of P ; (C) Absent P in lead I.

Q Wave.—Q was absent in a large number of cases and was never more than 4·5 mm. in amplitude.

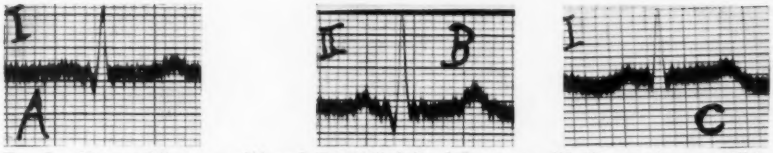

FIG. 2.—Types of Q wave : (A) and (B) Well-defined Q waves ; (C) Q scarcely perceptible.

R Wave.—The limits of variation in this wave were extreme. In lead I the wave might be as little as 1·5 mm. or as much as 18·6 mm. ; in lead II as little as 3·6 mm. or as much as 23·6 mm. ; whilst in lead III it varied between 0·5 and 20·5 mm. Slight notching of R was fairly common in all leads, especially in the descending limb. Splitting and low voltage of the QRS complex in lead III was very common.

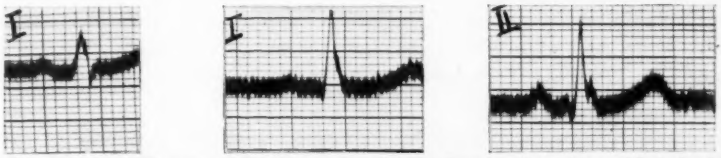

FIG. 3.—Types of R wave : Three examples of slight degrees of notching of R in lead I.

S Wave.—This varied from 0·0 to 11·5 mm. and Fig. 4 shows well-defined waves. In many cases the descending limb of R did not come straight down to the iso-electric line, but sloped gradually into it (Fig. 5).

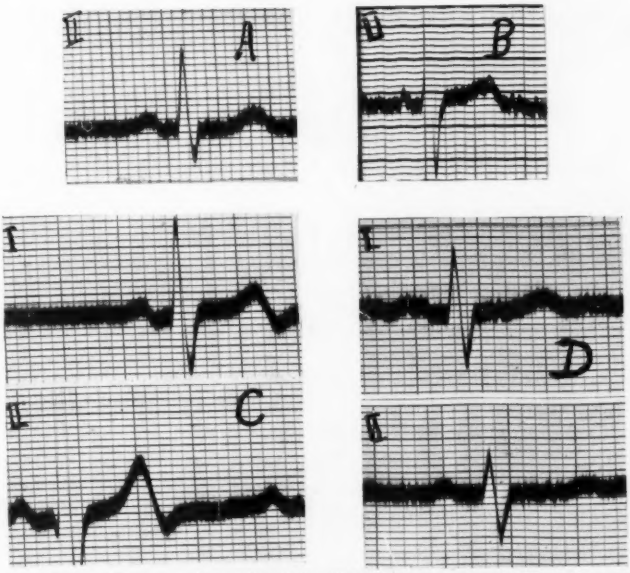

FIG. 4.—Types of S wave : (A) and (B) S well defined, prominent in B ; (C) and (D) S prominent leads I and II.

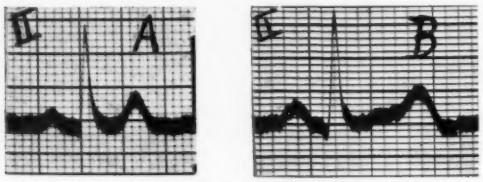

FIG. 5.—Types of S wave : (A) and (B) S indeterminate, slurring into R-T segment.

S-T Interval.—The S-T interval was studied with particular care in view of its great importance in relation to coronary thrombosis. Slurring of the R wave into the S-T segment was not uncommon (Fig. 2) especially when the portable electrocardiograph was used, and must also be distinguished from genuine S-T deviation. Out of 302 cases only 5 records showed the slightest S-T deviation. Occasionally there was a false impression of S-T deviation due to the fact that the P-Q interval sank below the iso-electric level. The degree of S-T deviation observed in the cases mentioned did not amount to more than 1·5 mm. (Fig. 6).

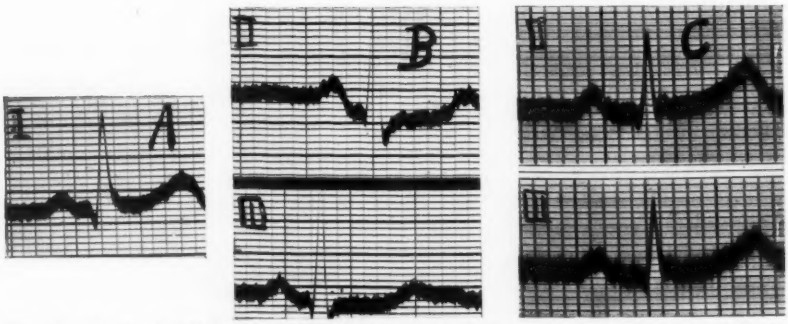

FIG. 6.—Types of S-T interval : (A) Slight S-T deviation ; (B) Slight S-T depression in leads II and III ; (C) False impression of S-T deviation owing to depression of P-Q interval.

The configuration of the S-T interval varied greatly in different normal subjects. In some there was a long iso-electric period often lasting 0·16 of a second, while in others it was shorter or absent (Fig. 7). In some cases where there was no iso-electric period, the T wave took origin in a steep fashion from a deep S wave (Fig. 8).

QRS Complex.—The QRS complex showed great variation in lead III. This fact is well established, and it is rare to find any pathological importance attached to it. A more careful study was made of the variation in leads I and II. Out of the 302 cases examined, only 15 showed any notching of the QRS in these leads (Fig. 9), and another 11 showed low voltage without notching (Fig. 10). Whenever notching was prominent some degree of low voltage was usually present.

Axis Deviation.—Right ventricular preponderance was noted in 3 cases in a pronounced form and to a slight degree in 15 others. Left ventricular

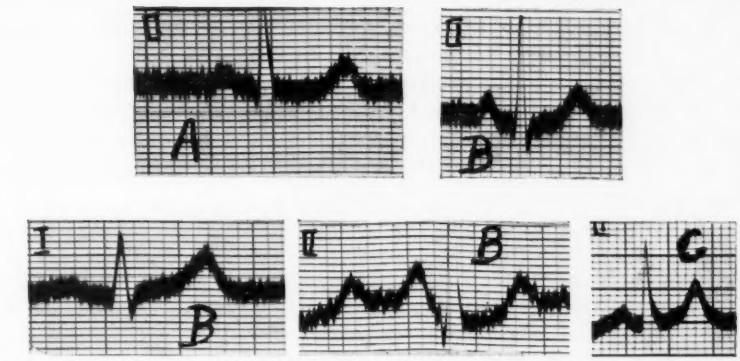

FIG. 7.—Type of S-T interval in leads I and II : (A) Long iso-electric period before T wave ; (B) Short iso-electric periods ; (C) No iso-electric period.

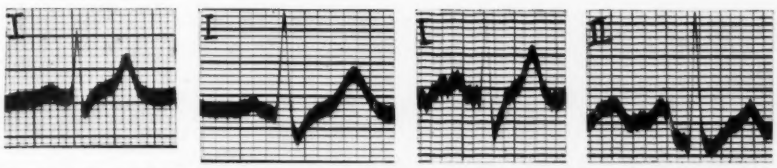

FIG. 8.—Types of S-T interval in leads I and II : Four cases showing no iso-electric period, T taking origin from a deep S wave.

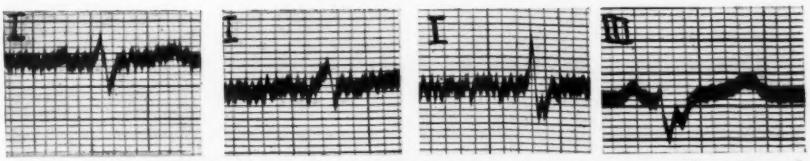

FIG. 9.—Types of QRS ; Notching : The first three cases show various types of notching of QRS in lead I ; the fourth shows pronounced notching of QRS in lead III.

FIG. 10.—Types of QRS ; Low voltage : Two examples of low voltage in lead I.

preponderance occurred in a moderate or pronounced form 26 times, i.e., in 8 per cent. of all cases; and slighter degrees were present in another 24 cases. In the third decade where 136 cases were available, there were 9 with slight and 7 with pronounced left ventricular preponderance, i.e. a total of 12 per cent. It would appear, therefore, that left axis deviation is a comparatively common finding in the normal electrocardiogram. It is notable that minor degrees of ventricular preponderance vary from complex to complex, probably due to respiratory variations.

T Wave.—The T wave varied greatly in amplitude, in the case of positive waves from 0·0 to 12·5 mm. The commonness of inversion of T 3 is universally recognized, but it is interesting to note that it occurred in as many as 87 out of 302 cases. In view of the attention paid to the various shapes of inverted T (e.g. in digitalis poisoning and coronary thrombosis) it is also important to recognize that considerable variation may be found in the normal from rounded waves to quite sharp ones (Fig. 11). In no case was inversion of T observed in lead I (although T was often flat), but inversion in leads II and III together was present in four cases.

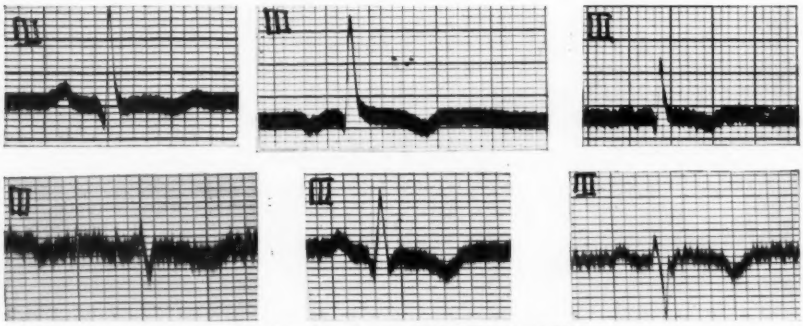

FIG. 11.—Types of negative T in lead III.

Time Intervals : P-R.—This interval varied from 0·12 to 0·22 of a second. The reading of 0·22 occurred in four subjects, three of whom were young people (Fig. 12). This is notable in view of the generally accepted view that the P-R interval should not exceed 0·20 of a second.

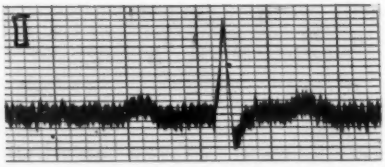

FIG. 12.—Prolonged P-R.: Four examples of a P-R interval of 0·22 of a second occurred in this series of 302 normal persons.

QRS.—The minimum was 0·04 seconds and the maximum 0·10, figures not varying from those usually accepted as normal.

S-T.—The minimum was 0·20 of a second, though such a low figure was only observed in a child. The maximum was 0·36 of a second.

Discussion of Results

Table I has been constructed to compare the average measurements of the electrocardiogram in the second, third, and fourth decades with those in the fifth, sixth, and seventh. As Lewis's work was principally on young persons, it will be more satisfactory to compare his results with those of the second, third, and fourth decades.

A comparison indicates that, on the whole, there are no great differences between his figures and those of the present series, though in the latter the amplitude of most waves appears to be rather greater.

The P wave showed an average amplitude of 0·8 and 1·9 mm. in leads I and II, compared with figures of 0·52 and 1·16 mm. in Lewis's series. In both series P averaged 0·8 mm. in lead III.

The measurements of Q were almost identical in the two series. The measurements of R were uniformly greater in all three leads in the present series. S showed no appreciable difference, and T was only slightly greater in leads I and II.

TABLE I

Comparison of Average Measurements in Normal Subjects

Decade	P	Q	R	S	T
Lead I					
2nd, 3rd, and 4th	0·8	0·5	8·1	2·0	2·8
5th, 6th, and 7th	0·7	0·7	7·9	1·5	2·2
Lead II					
2nd, 3rd, and 4th	1·9	0·8	14·0	3·0	3·2
5th, 6th, and 7th	1·8	1·1	11·4	2·4	2·8
Lead III					
2nd, 3rd, and 4th	0·8	0·6	8·8	1·5	0·3
5th, 6th, and 7th	0·8	0·7	5·8	2·1	0·0

Age Variations

The changes with age were less than might have been anticipated. There were no significant changes in the P waves in any leads.

The Q waves showed a slight increase in all leads in the aggregate of the fifth, sixth, and seventh decades.

The R waves in leads II and III appeared to be maximum in the third and fourth decades, but the amount of variation was not enough to be of any importance. On the whole, however, the later decades showed a rather smaller amplitude in the R waves than the earlier ones.

The S waves did not appear to be materially altered.

The T wave in lead I seemed to be larger in the earlier decades of life. In leads II and III it was also rather larger, though comparison of the average figures for the second, third, and fourth decades with the fifth, sixth, and seventh decades did not show any great difference.

Table II shows that there was no appreciable difference in the average P-R interval or in the duration of QRS or of S-T between the three earlier and the three later decades.

TABLE II

AVERAGE DURATION OF P-R, QRS, AND S-T IN LEAD II IN EARLIER AND LATER DECADES

	P-R	QRS	S-T
2nd, 3rd, and 4th decades ..	1·8	0·06	0·28
5th, 6th, and 7th decades	1·7	0·06	0·28

Left ventricular preponderance, as might be expected, was greater in the later decades of life. Out of 136 cases in the third decade it occurred in a pronounced form in 5 per cent., whilst out of 88 cases in the fifth, sixth, and seventh decades it occurred in 16 per cent. Those cases which showed inversion of T in leads II and III occurred in the third and fourth decades. The incidence of negative T waves in lead III was, curiously enough, greater in the first decade (6 cases), though the total number available in this decade was too small (8 cases) to draw any definite conclusions. Apart from this anomaly, the incidence of a negative T wave in lead III appeared to be roughly similar in all decades of life.

SUMMARY

1. To determine the limits of physiological variation in the electrocardiogram and any changes that might occur with age, 302 normal subjects have been investigated. Each was subjected to a rigorous examination, including blood pressure readings and Wassermann reaction. The following points are deemed worthy of emphasis.

2. The P wave was often flat or scarcely perceptible in lead I and occasionally had a split or broad summit. A few examples of inversion of P 3 were seen.

3. The R wave varied within wide limits in its amplitude and slight notching of the descending limb was not uncommon.

4. The S wave, although usually well defined, may slope gradually into the R-T segment.

5. True S-T deviation was so rare and so minute that it must be regarded with great suspicion, but peculiarities in the formation of the S-T interval were very common and must not be confused with genuine S-T deviation.

6. Slight degrees of notching and of low voltage in the QRS complex in leads I and II were not uncommon. The types of record found can be seen better by inspection of the illustrations than by description.

7. Right axis deviation in a pronounced form was uncommon even in young persons, whilst left axis deviation occurred quite frequently at all ages, increasingly so in the later decades where it was found in one sixth of all cases.

8. Inversion of T was never found in lead I, but inversion of T in both leads II and III was present in four cases. The former must be considered as pathological whenever it occurs, and the latter as suspicious. Inversion of T in lead III only was present in 29 per cent. of the total 302 cases and considerable variation in the shape of the wave was recognized.

9. The only unusual finding as regards time intervals was the occurrence of a few cases where the P-R interval was 0·22 of a second.

10. Age variations were slight, the most important being an increase in the left axis deviation in the later decades of life.

We wish to thank Dr. J. L. Chisnall for his help in the statistical part of this work and Mr. E. Caldwell for his careful work with the electrocardiograms.

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Lewis, T., and Gilder, M. D. D. (1912). *Phil. Trans. Roy. Soc., Lond.*, **202**, 351.

TABLE III

MEASUREMENTS OF P, Q, R, S, AND T WAVES IN NORMAL SUBJECTS

	P	Q	R	S	T
Lead I	First decade ; 8 cases				
Average	0·9	1·0	8·2	1·7	3·1
Maximum	1·6	3·6	11·6	7·6	4·6
Minimum	0·0	0·6	5·6	0·0	1·6
Lead II					
Average	1·5	0·5	12·5	1·7	2·2
Maximum	3·0	4·0	19·5	2·5	5·5
Minimum	0·5	0·0	7·5	0·5	0·5
Lead III					
Average	−0·2	0·4	4·1	0·8	−0·5
Maximum	1·5	3·5	10·5	1·5	1·5
Minimum	−0·5	0·0	1·5	0·5	−1·5

TABLE III—*continued*

MEASUREMENTS OF P, Q, R, S, AND T WAVES IN NORMAL SUBJECTS

	P	Q	R	S	T
Lead I		Second decade ; 25 cases			
Average	1·0	0·9	8·8	2·3	3·3
Maximum	2·5	3·5	18·6	8·0	4·5
Minimum	0·0	0·0	2·0	0·0	1·5
Lead II					
Average	1·7	1·1	11·8	2·6	3·0
Maximum	2·6	2·5	23·6	11·5	6·5
Minimum	0·5	0·0	6·6	0·0	1·6
Lead III					
Average	0·2	0·5	6·0	1·6	1·1
Maximum	2·5	4·5	14·4	8·5	3·5
Minimum	−4·4	0·0	1·5	1·5	−2·0
Lead I		Third decade ; 136 cases			
Average	0·6	0·4	9·0	1·6	3·1
Maximum	1·5	2·6	18·6	7·5	6·5
Minimum	0·0	0·0	2·5	0·0	1·1
Lead II					
Average	1·7	0·5	15·6	3·0	4·2
Maximum	2·7	2·5	23·5	10·6	12·5
Minimum	0·5	0·0	6·5	0·0	0·5
Lead III					
Average	0·7	0·5	9·3	1·5	−0·3
Maximum	2·5	3·6	20·5	8·5	6·5
Minimum	−3·5	0·0	1·5	0·0	−5·5
Lead I		Fourth decade ; 36 cases			
Average	0·8	0·1	6·4	2·2	2·1
Maximum	1·5	0·5	10·5	4·5	3·6
Minimum	0·0	0·0	2·0	0·5	1·5
Lead II					
Average	2·4	0·7	14·6	3·5	2·4
Maximum	5·0	1·5	18·5	8·5	6·6
Minimum	0·5	0·5	7·5	0·5	−3·5
Lead III					
Average	1·5	0·9	11·2	1·5	0·1
Maximum	3·0	3·0	18·5	8·6	4·6
Minimum	−0·5	0·0	4·5	1·5	−4·5
Lead I		Fifth decade ; 42 cases			
Average	1·0	0·1	7·5	1·9	2·5
Maximum	2·6	2·0	13·5	3·6	3·5
Minimum	0·5	0·0	2·6	0·0	0·6
Lead II					
Average	2·0	0·6	12·4	3·0	2·7
Maximum	2·6	2·6	16·4	5·0	4·6
Minimum	0·0	0·0	4·5	0·0	0·6
Lead III					
Average	1·0	0·4	6·8	2·2	0·0
Maximum	2·6	4·0	13·4	4·5	2·5
Minimum	−4·0	0·0	1·6	0·0	−5·0

TABLE III—*continued*

MEASUREMENTS OF P, Q, R, S, AND T WAVES IN NORMAL SUBJECTS

	P	Q	R	S	T
Sixth decade ; 33 cases					
Lead I					
Average	0·6	0·7	7·8	1·2	2·0
Maximum	1·5	2·5	13·6	6·5	3·5
Minimum	0·0	0·0	1·5	1·5	0·0
Lead II					
Average	1·9	1·3	11·3	2·3	3·0
Maximum	3·5	4·5	16·5	9·5	7·5
Minimum	0·5	0·0	5·5	1·5	0·5
Lead III					
Average	1·4	1·2	5·9	2·2	0·5
Maximum	3·0	4·5	14·6	10·5	4·5
Minimum	−1·5	0·0	0·0	1·5	−4·5
Seventh decade ; 13 cases					
Lead I					
Average	0·9	1·2	8·4	1·4	2·1
Maximum	1·5	2·5	13·6	2·5	3·5
Minimum	0·0	0·0	2·5	0·5	0·5
Lead II					
Average	1·5	1·5	10·4	2·0	2·8
Maximum	2·5	3·0	17·6	4·5	5·5
Minimum	0·5	0·0	3·6	0·0	1·5
Lead III					
Average	0·2	0·5	4·6	2·0	−0·7
Maximum	1·5	2·5	18·6	7·5	3·5
Minimum	−1·5	0·0	0·5	0·0	−4·5

TABLE IV

MEASUREMENTS OF P-R, QRS, AND S-T INTERVALS IN SECONDS, IN NORMAL SUBJECTS (LEAD II ONLY)

	P-R	QRS	S-T
First decade			
Average	0·14	0·05	0·25
Maximum	0·16	0·06	0·28
Minimum	0·12	0·04	0·20
Second decade			
Average	0·16	0·06	0·28
Maximum	0·22	0·08	0·32
Minimum	0·14	0·04	0·24
Third decade			
Average	0·19	0·06	0·29
Maximum	0·22	0·08	0·36
Minimum	0·14	0·04	0·24
Fourth decade			
Average	0·18	0·06	0·28
Maximum	0·22	0·08	0·32
Minimum	0·14	0·04	0·24
Fifth decade			
Average	0·18	0·05	0·29
Maximum	0·20	0·06	0·34
Minimum	0·14	0·04	0·24
Sixth decade			
Average	0·17	0·06	0·28
Maximum	0·20	0·10	0·32
Minimum	0·14	0·04	0·22
Seventh decade			
Average	0·17	0·06	0·28
Maximum	0·20	0·08	0·32
Minimum	0·14	0·04	0·24

PERSISTENT CONDUCTION DEFECTS FOLLOWING DIPHTHERIA

BY

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Received December 29, 1938

Whether diphtheria commonly causes any permanent cardiac damage has long been the subject of discussion; but it is generally accepted that the persistence of electrocardiographic changes of any serious significance is extremely rare (Alstead, 1933 ; Beer, 1936 ; Begg, 1937 ; Hoskin, 1926 ; Jones and White, 1927 ; and Thompson, Golden, and White, 1937). It is not uncommon for complete heart block and bundle branch block to develop during the course of severe diphtheria, usually during the second week (Alstead, 1932 ; Begg, 1937 ; Franck, 1935 ; Place, 1932 ; and Schwensen, 1922). The mortality amongst cases showing these changes is high, but if the patient survives there is usually a complete cardiac recovery.

Parkinson (1915) has described the case of a man, aged 22, who, on the twenty-second day of a severe attack of diphtheria, developed complete heart block. This lasted for eleven days, when auricular fibrillation supervened. At first the ventricular rate was slow (50 per minute), but later increased to 100–110 per minute. The fibrillation persisted for at least six months. Sprague and White (1927) have reported the case of a woman, aged 22, with complete heart block dating from diphtheria 19 years before. Read (1929) described a woman, aged 48, with complete heart block which, it was believed, dated from an attack of diphtheria at the age of 6; and Chamberlain and Alstead (1931) have published the details of a woman, aged 28, with complete heart block following diphtheria 18 years before. Butler and Levine (1930), investigating twenty cases with heart block of obscure origin, found a history of diphtheria in ten, whereas of 600 surgical cases studied as controls the incidence of a history of diphtheria was only six per cent. As a result of these observations it was suggested that diphtheria may predispose to the development of heart block in later life. However, Thompson, Golden, and White (1937) investigated ninety-one patients who had severe diphtheria 15 to 20 years previously, but found no evidence that diphtheria predisposed to the subsequent development of conduction defects.

In view of the rarity of persistent defects in cardiac conduction after recovery from diphtheria, the three following cases appear worthy of record. In two there was persistent complete heart block and in one persistent bundle branch block.

Two Cases of Complete Heart Block.

Case 1. *J. H., female, aged* 14. *Reported previously* (*Perry*, 1931).

On November 13, 1929, this patient was admitted to Ham Green Fever Hospital on the third day of a severe attack of diphtheria. There was extensive membrane and a " bull neck." She was given 40,000 units of anti-toxin. On November 19 the pulse dropped to 70 and on December 10 to 54–60 per minute. From this time until she was discharged on February 6, 1930, the pulse rate varied from 48–64. Palatal palsy necessitated nasal feeding for a time. Complete heart block was first demonstrated on September 30, 1930, and since that time there has been no change in the cardiac rhythm or in the physical signs.

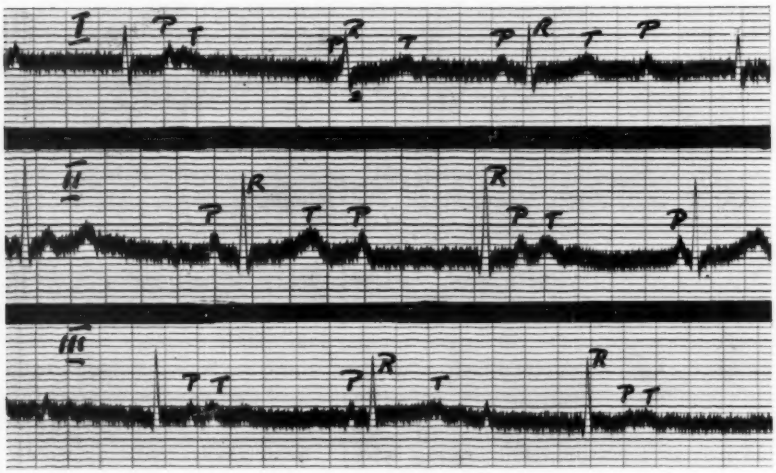

Fig. 1.—Electrocardiogram of Case 1.

She attends school normally, but is not allowed to take part in games or drill. There have been no symptoms referable to the cardiac lesion. The apex beat is diffuse and forcible in and beyond the mid-clavicular line. The heart sounds vary in intensity from beat to beat and there is an inconstant diastolic sound. There are no murmurs. The blood pressure is 115/70. An X-ray shows a heart which is generally enlarged, particularly the left ventricle, with a cardio-thoracic ratio of 65. An electrocardiogram shows complete heart block with a ventricular rate of 50.

Case 2. *J. T., female, aged* 13.

This patient was admitted to Ham Green Hospital on June 27, 1931, with very severe diphtheria which had commenced on June 23. There was extensive

membrane and œdema of the neck. She received 46,000 units of anti-toxin intravenously with five ounces of 20 per cent. glucose. At the end of the second week she showed the cardiac vomiting syndrome, which was treated by intravenous glucose and finally stopped. On July 8 palatal paralysis developed, followed on July 31 by strabismus and ptosis. After the second week the pulse remained between 80 and 90 until September 2, when it fell to 60. At the same time there was vomiting, pyrexia, and slight enlargement of the neck glands. The patient was discharged from hospital on September 18. On October 13 an electrocardiogram showed complete heart block. At that time there was some weakness of the leg muscles.

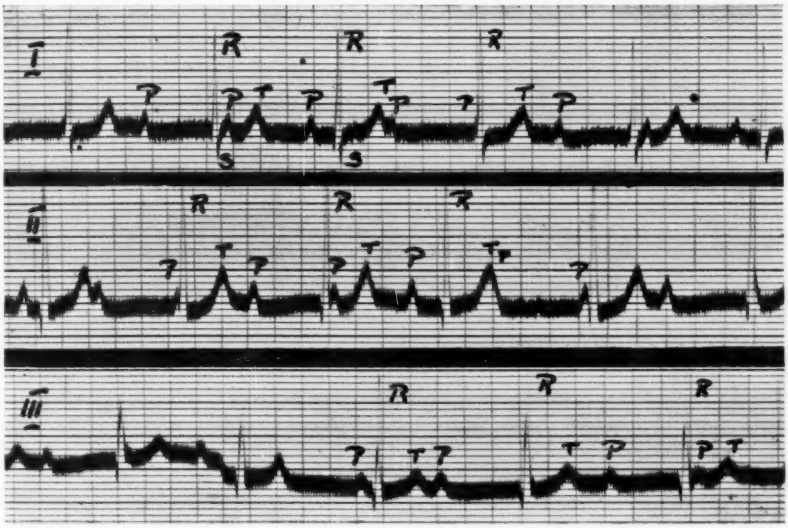

FIG. 2.—Electrocardiogram of Case 2.

By April 1932 she appeared to be perfectly well, but the cardiac condition was unchanged and has remained so ever since. She attends school normally, but is not allowed to take part in games or drill. The apex beat is diffuse and forcible beyond the mid-clavicular line. The heart sounds vary in intensity from beat to beat and there is an inconstant mid-diastolic sound. There is a loud apical systolic murmur. The blood pressure is 120/65. An X-ray shows an enlarged left ventricle with a cardio-thoracic ratio of 56, and a prominent pulmonary artery. The electrocardiogram shows complete heart block with a ventricular rate of 59.

Comment.

In these two cases of persistent heart block the conduction defect dates from an attack of severe diphtheria. In the first the heart block developed at the

usual time, during the second week of the illness. The very late development of the conduction defect in the second, associated with an apparent relapse or fresh infection forty days after the onset of the disease, suggests that the heart block may possibly be due to an attack of rheumatic carditis, and the apical systolic murmur may be considered further evidence of this. However, no other manifestations of acute rheumatism were noted and there has been no rheumatic episode since the heart block was discovered, such as might have been expected had this been rheumatic in origin. Alstead (1932) found that in the acute stage of diphtheria complete heart block might easily pass unnoticed without electrocardiographic studies, since the ventricular rate may be as high as 100 per minute. In view of this it is possible that the heart block in the second case developed before September 2, but this seems rather unlikely since there was a definite setback at the time with pyrexia and vomiting, quite apart from the fall in pulse rate.

A feature of both cases is the comparatively rapid ventricular rate. A similar rate has been noted in children with complete heart block due to rheumatic heart disease (Perry, 1931). It is interesting to note that in the cases of diphtheritic heart block described by Chamberlain and Alstead (1931) and by Read (1929) the pulse rates were both over 40 at the age of 28 and 48 respectively. It is curious that these two cases and the three reported by Sprague and White, by Chamberlain and Alstead, and by Read are all females.

One Case of Bundle Branch Block.

Case 3. *G. H., male, aged* 16.

This boy was admitted to Ham Green Hospital on January 10, 1931, on the third day of a severe attack of diphtheria with extensive membrane and œdema

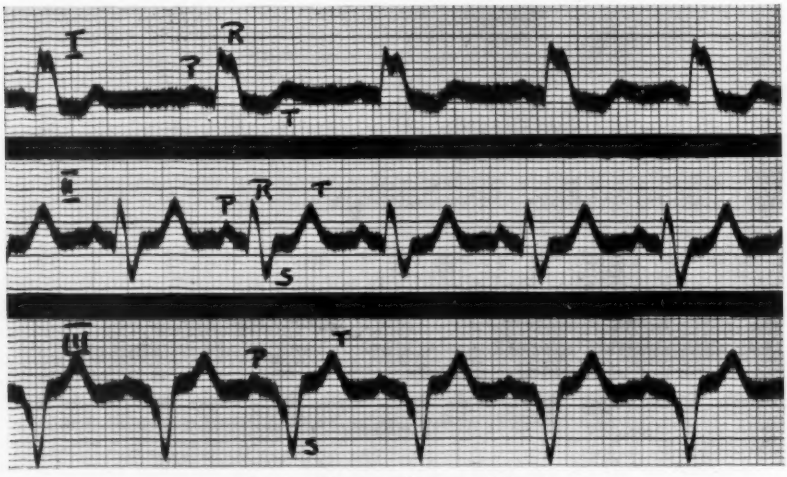

Fig. 3.—Electrocardiogram of Case 3

of the neck. He received 40,000 units of anti-toxin intravenously. On January 18 he developed palatal paralysis. On February 3 he vomited, the heart was dilated, and the liver was enlarged and tender. He was treated by intravenous glucose and improved. The heart remained dilated and the pulse rate 104–112 until April 29, when he was discharged from hospital at the parents' request.

On March 16, 1932, he was quite well and had no symptoms. The apex beat and heart sounds were normal and there were no murmurs. An electrocardiogram showed bundle branch block of the common (left) variety; this has been present whenever he has been examined since. At times a soft apical systolic murmur has been noted, but this is not constant. The blood pressure was 110/65. An X-ray showed a small vertical heart with a cardio-thoracic ratio of 45. He attended school normally, but was not allowed to play games or to do drill. He is now (1938) working quite successfully.

Comment

This last patient obviously suffered from very severe cardiac damage during the attack of diphtheria, and it would appear justifiable to attribute the persistent bundle branch block to this, since there has been no suggestion of any rheumatic infection before or after. This would seem to be the first record of a case of bundle branch block due to diphtheria that has persisted with the recovery of the patient.

Summary

A description has been given of three children who recovered from attacks of diphtheria with persistent conduction defects. In two this took the form of complete heart block and in the third of bundle branch block. In all three the lesion seems to have developed at the time of their diphtheria or shortly after, and in all it has persisted for some years.

I am greatly indebted to Dr. B. A. Peters for very kindly supplying the information about these cases while they were in the Fever Hospital.

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PAROXYSMAL TACHYCARDIA ; ÆTIOLOGY AND PROGNOSIS OF ONE HUNDRED CASES

BY

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The early papers on paroxysmal tachycardia gave its essential features with great accuracy, and the modern reader will enjoy their lucid descriptions. After the classic account by Cotton (1867) with pulse tracings by the great Burdon-Sanderson, many cases were reported, and Bristowe (1887) and Bouveret (1889) collected these and added more of their own. Bristowe's paper was " On recurrent palpitations of extreme rapidity in persons otherwise apparently healthy " and Bouveret first introduced the modern name in " De la Tachycardie essentielle paroxystique " ; he stressed that it did not produce organic heart disease, but thought it a serious malady, and as he recorded a fatal outcome in many of the longer attacks he may be responsible for the guarded prognosis often given in books. A few of these attacks were perhaps paroxysmal flutter, but with this exception the papers just quoted might still be read as accurate accounts of much of our clinical knowledge. Herringham (1897) emphasized that paroxysms often started in childhood and that the patient lived to a good age. Hoffmann (1900) wrote a book analysing 135 reported cases.

For some time little more was added, till the electrocardiogram made our knowledge more precise and enabled us to distinguish paroxysmal auricular flutter and to differentiate the various types of paroxysmal tachycardia. Hume (1930) has reviewed the whole subject, giving a full list of references, and for this reason most papers are only referred to here in connection with special points as they arise. Many of the large number of recent papers deal with the electrocardiographic features of single cases and pay less attention to the clinical picture of paroxysmal tachycardia as a whole.

This paper is based on observations on one hundred cases seen during the years 1925–32. A few with no features of special interest may have escaped indexing, but they were not many, and otherwise it is an unselected series. Cardiograms * of the paroxysms were obtained in 42 ; 30 others were seen by

* A good short word has not yet been coined for electrocardiogram, which is wasteful of time and space now it is in such constant use ; no confusion is likely to arise by the substitution of the word cardiogram.

a doctor (occasionally by one of us) in the attack, though no graphic record was obtained ; and in 28 the diagnosis was made on the history alone. The occurrence of paroxysms in five students at Guy's Hospital during the years 1928–30 suggested that the condition was not uncommon, but only two more have been seen in the subsequent seven years.

Many of these patients have been followed over a long period, and as the immediate prognosis is nearly always good, this is essential for any complete picture. We have followed 16 until their death, not always from heart disease ; 53 have been seen or have replied fully to letters in 1937–38 and most of these and another 13 were seen in 1934–35, and we have no reason to expect any great change in their condition. The remaining 18 were last seen during 1925–32 and generally attacks had already occurred for some years. The series is therefore likely to give a fair view of those cases severe enough to come under medical observation, and has been followed long enough to give some approximation to a correct prognosis.

DIAGNOSIS

Often the attack is not seen and there is only the history ; this applied to 28 of our patients. Reliance must then be placed on the abrupt onset, and as a rule on the equally abrupt end. The rapid beating of the heart is nearly always the first symptom felt, whatever others may follow. If the patient is able to define the number of attacks and their duration it is almost diagnostic, since ordinary palpitation is much more vague and indefinite in its character. Enquiry must then be made as to whether the heart was regular or irregular, and, perhaps surprisingly, the patient's statement usually proves to be reliable, and only one had to be excluded from this series when a subsequent record showed it to be paroxysmal fibrillation. This suddenness of onset is the most reliable single symptom for diagnosis—" As if the pendulum of a clock had suddenly lost its weight " in the words of one patient. During the attack the heart action is a constant steady fluttering, and this word is often used.

The end of the attack is usually as sudden as the onset, but when the heart is diseased the patient may often be incapable of saying exactly when the end came. " I think it ended a few minutes ago, but it is still going very fast " was one description. This is specially so when the heart muscle is becoming worse, and in one such observation the rate appeared to fall gradually from 180 to 100 in the course of three hours, simple tachycardia having followed the paroxysm so that the exact ending was difficult to detect clinically : the offset is therefore less reliable than the onset. Most other symptoms are too variable to be reliable, and extrasystoles before or after the attack may obscure the clear picture.

When the attack is seen the diagnosis rarely remains doubtful, though it may not be thought of if the heart rate is 140 or less. It is sometimes difficult to distinguish paroxysms of tachycardia from those of flutter if a constant degree of heart block makes the ventricular rate regular, and impossible if the diagnosis has to be made on the history alone. The auricular rate in flutter is

generally 240–360; 4 : 1 heart block is rare in an untreated patient, and regular 3 : 1 heart block extremely rare; but 2 : 1 heart block is common and gives a rate of 120–180, i.e. within the usual limits of paroxysmal tachycardia. Again, flutter with the auricle at a slow rate without heart block (220–240) may be difficult or impossible to distinguish from paroxysmal tachycardia even on the cardiogram, and the decision may have to be made on clinical grounds. Lastly, a paroxysm of tachycardia may fall above its usual limits of 240 (Fig. 1).

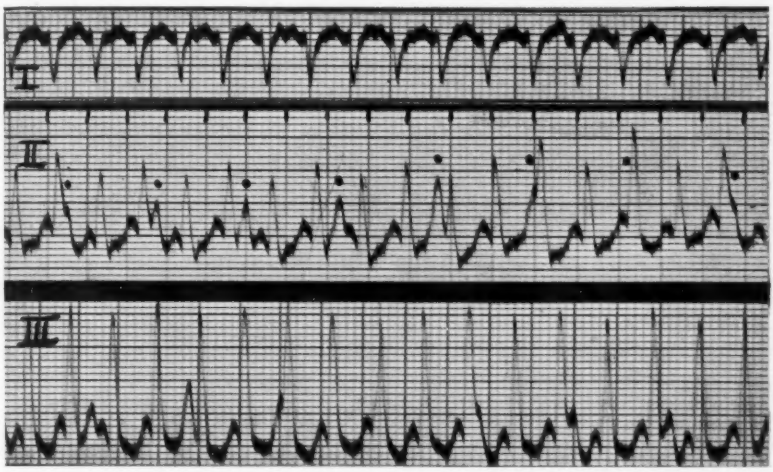

FIG. 1.—Ventricular tachycardia, auricle 128, ventricle 274; from a boy whose heart was otherwise normal (Case 69). The auricular waves have been marked with a ● in lead II. The time marker in this and subsequent records indicates fifths and twenty-fifths of a second.

Apart from the rate, the following points may help to distinguish between paroxysmal flutter and tachycardia. If the regular rhythm is occasionally interrupted by a slower or irregular rhythm; if the rate can be halved by pressure on the neck; or if in the longer attacks the rate can be halved or made irregular with digitalis, the attack is flutter. Flutter, like fibrillation, is more often established than paroxysmal, but Parkinson and Bedford (1927) found that nearly one quarter of their cases of flutter had the paroxysmal form. Paroxysmal tachycardia very rarely lasts more than ten days and generally not more than four days: therefore the longer an attack of unknown origin lasts, the more likely it is to be flutter, and after ten days this becomes more certain. If the patient has high blood pressure or arteriosclerosis or if congestive failure develops rapidly, flutter must be thought of but cannot be diagnosed merely on these grounds. Occasionally even when a cardiogram is obtained there may still be difficulties, e.g. slow flutter without heart block, or a faster rate with 2 : 1 heart block where the P and T waves cannot be clearly distinguished.

Types of Paroxysmal Tachycardia

Every effort should be made to obtain a cardiogram of an attack ; not only to differentiate flutter, but to decide the variety of paroxysmal tachycardia, which cannot be determined in any other way and is of value in prognosis. It may still be difficult or impossible to subdivide the supraventricular into the various types of auricular and nodal, but fortunately this seems of less practical importance.

Of the 42 paroxysms recorded, 8 were ventricular. In three it was possible to identify P, occurring regularly at a slower rate than the ventricular complexes and quite unrelated to them, and so to prove that the paroxysms were ventricular ; the auricular rates were 150, 130, and 100, and the ventricular 220, 272, and 166 respectively (Fig. 1). In the remaining five ventricular records it was not possible to identify P, or there was retrograde conduction from the ventricle (Fig. 2). The type shown in Fig. 2 of very short paroxysms,

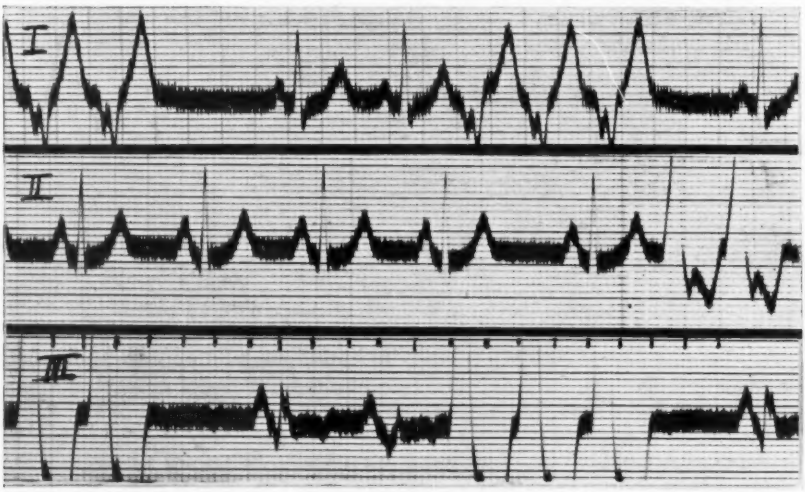

Fig. 2.—Short paroxysms of ventricular tachycardia, rate 142, probably with retrograde conduction ; from a young woman whose heart was otherwise normal (Case 34). This type may occur for years, apparently with little significance.

recurring frequently, may be found with a normal heart and seems to be of no serious significance even though it is ventricular.

If the QRS wave in the paroxysm was not widened or grossly aberrant, the focus was counted supraventricular. If QRS was widened and aberrant, it was compared with the QRS in the free intervals, and if different in conformation we considered the focus ventricular. Fig. 3 is an example we have called ventricular (though the P waves could not be seen) because the ventricular complexes of the attack differ from those of normal rhythm. This evidence is not conclusive, for the paroxysm may sometimes be auricular with aberrant

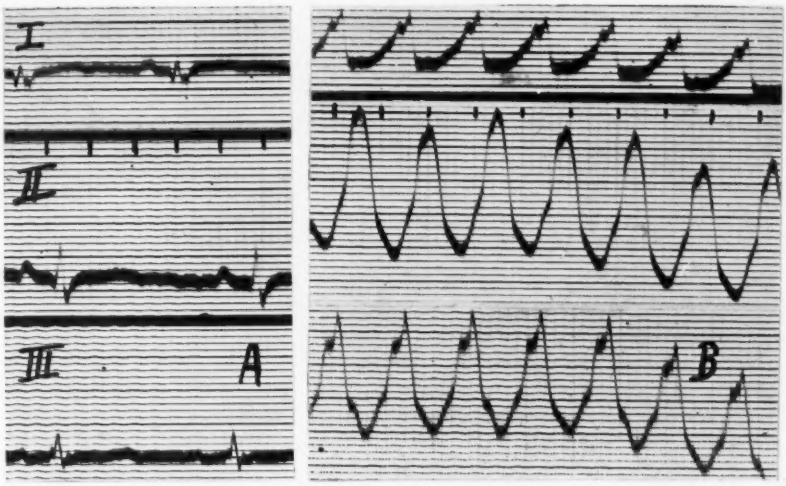

FIG. 3.—(A) Normal rhythm, and, (B) two days later, ventricular tachycardia, rate 207; there is no clear evidence of auricular activity but a suggestion of possible P waves, low on each second ventricular downstroke in lead III (Case 5).

conduction to the ventricle. If, on the other hand, QRS in the free interval was similar to that in the attack, we considered the focus as supraventricular. Such a paroxysm is shown in Fig. 4; it appears ventricular, but the QRS

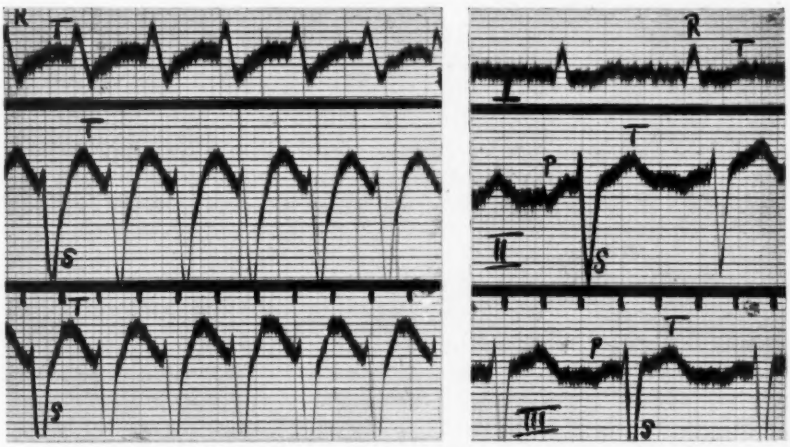

FIG. 4.—Supraventricular tachycardia, rate 175, simulating ventricular tachycardia because of the permanent defect of conduction: and normal rhythm; both from an elderly man with myocardial disease (Case 38).

complexes in the free intervals and in the attacks are identical; further, ventricular extrasystoles during normal rhythm did not resemble the ventricular waves of the paroxysm.

Levine (1927–28) has drawn attention to the slight irregularity in the rate and sounds in some ventricular paroxysms which may suggest the diagnosis on clinical grounds alone. On measuring the cardiograms this irregularity was present in two of our cases, but too slight to be appreciable to the finger or ear. Any gross irregularity is likely to indicate paroxysmal flutter with changing degrees of block.

Paroxysms of nodal tachycardia can usually be differentiated, but in many supraventricular paroxysms the exact site of origin cannot be defined. An example of auricular paroxysmal tachycardia is shown in Fig. 5. On the

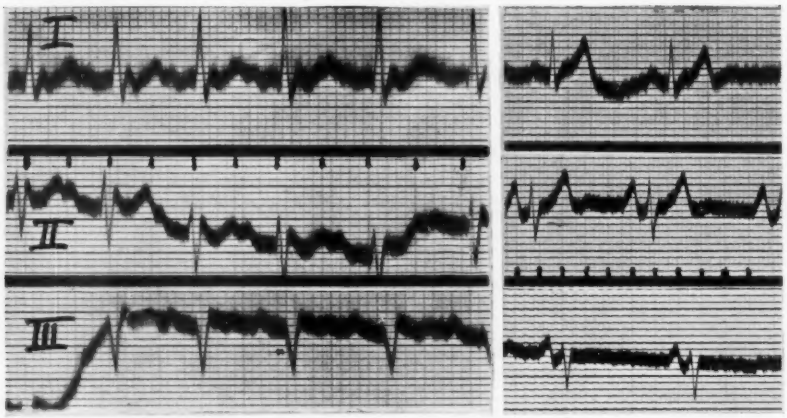

FIG. 5.—Auricular tachycardia, rate 142, simulating simple tachycardia : and normal rhythm ; both from an elderly man whose heart was otherwise normal (Case 98).

picture alone it could not be distinguished from simple tachycardia ; if the rate were a little faster P would be hidden in T, and one could not decide if it was an auricular attack with P hidden in T, or a nodal attack with P hidden in QRS. This seems to be a common difficulty when the beginning or ending of the attack is not seen (Figs. 9, 10).

Inversion of P indicates an abnormal focus in or near the A-V node, and its relation to QRS—whether before it with a short P-R interval, incorporated with it, or following it—indicates the level in the node of such focus. In two patients where the position and conformation of the P wave showed that the attack was nodal, it was inverted and preceded the QRS with a P-R interval that was normal (Fig. 6) ; this is sometimes described as superior or high nodal. In three more it was just before or with the QRS and was located by its large size (Fig. 7) or by recording a complete attack (Fig. 15a). This nodal group is relatively large in some published series and evidently includes many that we have left unclassified. The P wave was inverted and followed QRS in eight (Fig. 8), and these have been grouped as inferior or low nodal. In 13 of our 34 supraventricular cases there was no doubt of their nodal origin, two being high nodal, three nodal, and eight low nodal, and another three were

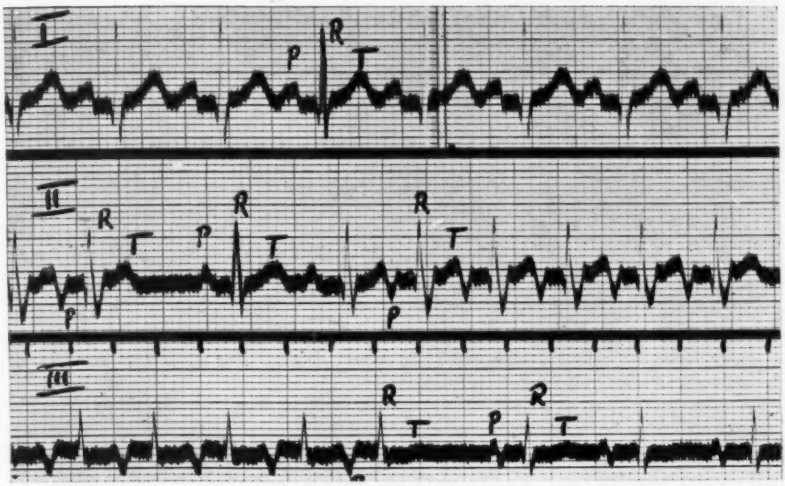

FIG. 6.—Nodal tachycardia of the " high nodal " type, rate 169, with the rhythm constantly changing ; from a boy whose heart was otherwise normal (Case 2).

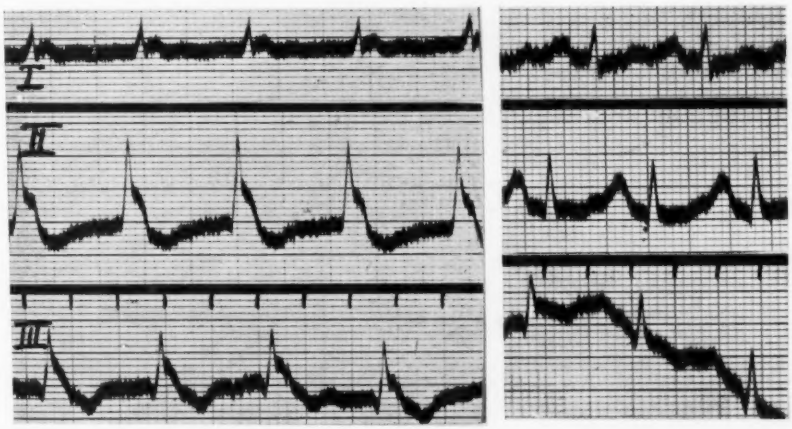

FIG. 7.—Nodal tachycardia of " intermediate " type, rate 125: and normal rhythm ; both from a woman with mitral stenosis (Case 87). The large P wave is incorporated with QRS on the downstroke of R.

probably nodal. This left 21 other supraventricular ; three were auricular but in the others there were no clear indications of the type (Figs. 9, 10).

Ritchie (1926) reviewed 138 cases, adding 14 of his own, and found one-third were ventricular. Hume (1930) collected the classification from various series as follows : auricular 40 ; superior nodal 8 ; inferior nodal 18 ; nodal 100 ; unclassified supraventricular 32 ; ventricular 55. But this is merely a

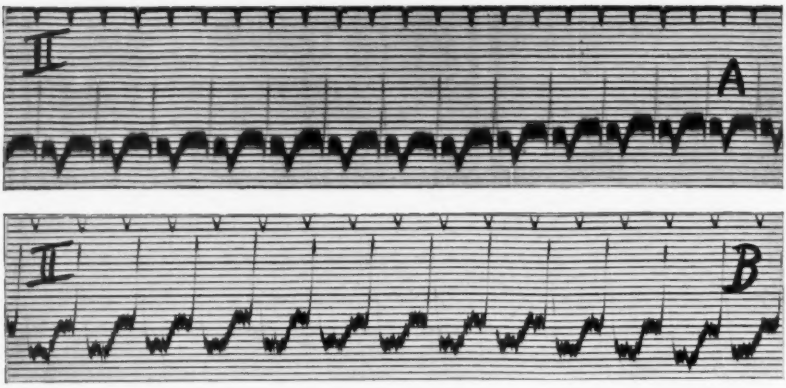

FIG. 8.—Nodal tachycardia of "low nodal" type, lead II only: (A) from a woman with mitral stenosis, rate 171 (Case 52); (B) from a woman with rheumatic aortic incompetence, rate 234 (Case 70).

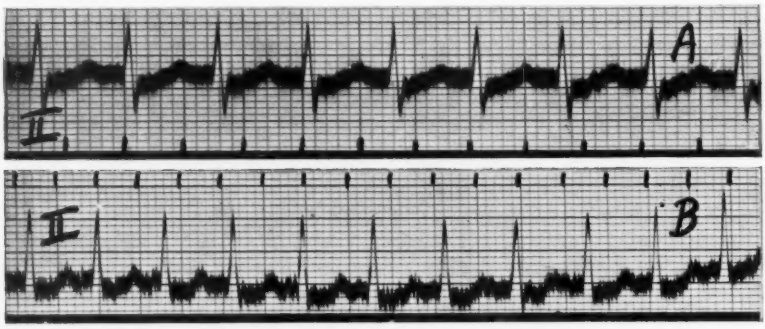

FIG. 9.—Supraventricular tachycardia, from two women with mitral stenosis: (A) rate 199 (Case 36); (B) rate 185 (Case 60).

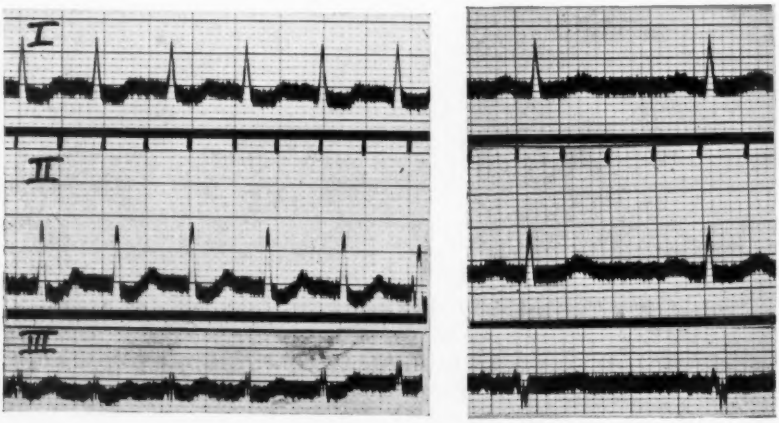

FIG. 10.—Supraventricular tachycardia, rate 172: and normal rhythm; both from an elderly man whose heart was otherwise normal (Case 58).

summary of some very discordant series, and we think that many classed as auricular or nodal should be left as unclassified supraventricular. The proportion of ventricular attacks in these series of Hume and Ritchie is in our opinion much higher than is an unselected series. Ventricular paroxysms occur most often in patients admitted to hospital for heart disease or even for failure, and so tend to be observed and reported ; many of these patients have been treated with digitalis and have had advanced heart disease ; and the paroxysms have occurred under these conditions on a few occasions, rather than periodically over a long time as with ordinary paroxysmal tachycardia. As instances, three of the five attacks reported by Gilchrist (1925–26) were almost terminal, and of the 65 patients reported by Strauss (1930) more than half were under treatment with digitalis for their heart failure and four-fifths had died within three months.

TABLE I

THE CORRELATION OF TYPES OF EXTRASYSTOLES AND OF PAROXYSMAL TACHYCARDIA

Type of Paroxysmal Tachycardia	Type of Extrasystoles recorded in the free intervals		
	Supraventricular only	Supraventricular and ventricular	Ventricular only
Supraventricular (34 cases)	5	5	2
Ventricular (8 cases) ..	0	0	4

It is said that the type of extrasystole observed in the free intervals will indicate the type of paroxysm and that extrasystoles are frequently observed in most cases of paroxysmal tachycardia. Often, however, this is not so, and as an example, in one patient seen each few weeks for ten years no extrasystoles have been noted. They were recorded in 16 of the 42 with records of their paroxysms (see Table I). Where supraventricular extrasystoles were observed (whether alone or with ventricular extrasystoles) the paroxysms were always of the supraventricular type, though auricular or nodal extrasystoles did not always indicate that the paroxysms were from the same focus. But where ventricular extrasystoles were observed the converse did not hold true : if they were the only type observed the paroxysms might be ventricular or supraventricular, though more likely to be the former ; but if they were of both types the paroxysms were always supraventricular.

ÆTIOLOGY

The first question to decide in each patient with paroxysmal tachycardia is whether there is any organic heart disease. This is essential for prognosis, and even slight signs may be very important. The ætiology of our hundred cases is shown in Table II, and rather more than half had no evidence of heart disease. The inclusion of those where there was no graphic record of the attack does not invalidate these conclusions about the ætiology ; the only

difference was that fewer records were obtained of the normals because they had less reason for attending hospital regularly.

TABLE II

ÆTIOLOGY OF 100 CASES OF PAROXYSMAL TACHYCARDIA

Ætiological Group	Number of Cases			
	Males	Females	Total	Number with Cardiogram during Attack
Normal	34	21	55	18
Rheumatic	4	15	19	13
Syphilitic	2	0	2	1
Hyperpietic	3	5	8	2
Thyro-toxic	0	4	4	1
Myocardial	9	3	12	7
Total	52	48	100	42

Men and women were equally affected, as the excess of women in the rheumatic group was balanced by the men in the myocardial and normal groups. The age incidence—the age at which the attacks started—is shown in Table III and Figure 11. In 11 per cent. they started before the patient was

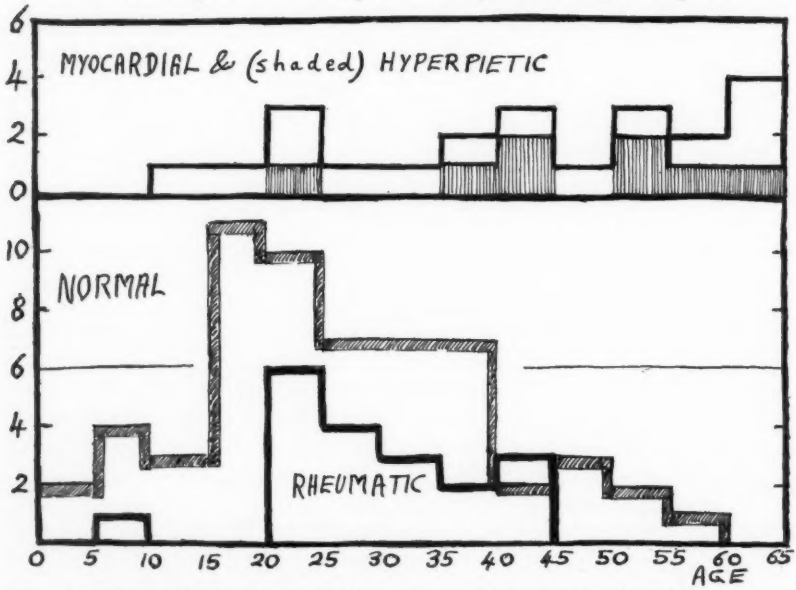

FIG. 11.—Diagram showing the age incidence at onset of paroxysmal tachycardia.

Above : 1. Hyperpietic and myocardial groups combined ; the hyperpietic are shaded and are mostly between 35 and 65, the myocardial are more evenly spread at all ages.

Below : 2. Normal group, at all ages with the main incidence between 15 and 40.

3. Rheumatic group, with the main incidence between 20 and 45 years of age.

fifteen, in 65 per cent. between fifteen and thirty-nine, and in another 12 per cent. before fifty, making 88 per cent. where the attacks started before this age.

TABLE III

AGE INCIDENCE AT ONSET OF PAROXYSMS

Age—in years	Number of Cases in each Group				Total
	Normal	Rheumatic	Hyperpietic	Myocardial	
0–4	2	—	—	—	2
5–9	4	1	—	—	5
10–14	3	—	—	1	4
15–19	11	—	—	1	12
20–24	10	6	1	2	19
25–29	7	4	—	1	12
30–34	7	3	—	1	11
35–39	7	2	1	1	11
40–44	2	3	2	1	7
45–49	3	—	—	1	5
50–54	2	—	2	1	5
55–59	1	—	1	1	3
60–	—	—	1	3	4
All ages ..	59*	19	8	14	100

* Includes 4 with goitre.

Most were hospital patients, and in those seen in practice since completing this series there have been fewer in the rheumatic group and more elderly patients with myocardial and coronary disease. They have been classified in the following six groups.

1. *Rheumatic Group :* 19 *cases.*—In all but one of these the attacks started between 20 and 44 years of age. There was valvular disease in 17, 10 with mitral, 5 with mitral and aortic, and 2 with aortic lesions ; the other 2 had a rheumatic history only.

2. *Syphilitic Group :* 2 *cases.*—Both had aortic incompetence ; and the paroxysms did not seem to influence the natural course of their disease. As there were no special features they have been included in the myocardial group for the tables, etc.

3. *Hyperpietic Group :* 8 *cases.*—Five of these, with an average age of 62 and an average blood pressure 180/105, may be taken as typical of the group. Their attacks were of relatively recent onset. Two elderly men were included, but probably the raised pressure was incidental, as their attacks had been present for 15 and 20 years ; two were not included, as paroxysms had occurred for 40 years. The eighth was unusual, as her attacks and high blood pressure both started after the removal of cystic ovaries. A less strict standard might have added one or two to this group, but among the normals there were very few who were even on the borderline.

4. *Myocardial Group :* 12 *cases.*—In many ways this was the most important group, though rather mixed. It comprised all those with heart disease who

could not be included elsewhere. Generally the signs were unmistakable, but occasionally even in young people the course of events showed serious heart disease with the paroxysms as the only early symptom, with a slight cardiac enlargement and/or an abnormal cardiogram as the only sign. They are discussed in more detail in the section on prognosis.

5. *Thyrotoxic Group : 4 cases.*—All were women with a goitre, but none had clinical hyperthyroidism. All have done well, and as they had no heart disease are included for subsequent discussion with the normal group. It is surprising that toxic goitre should be so much more frequently the cause of paroxysmal fibrillation (15 per cent. ; Parkinson and Campbell, 1930) than of paroxysmal tachycardia.

6. *Normal Group : 55 cases.*—The presence of paroxysms is not evidence that there is organic heart disease, and this group includes all those with hearts that were thought to be normal except for the paroxysms. Examples are given in the section on prognosis (see p. 149) of paroxysms for twenty or even sixty years without demonstrable heart disease at the end. Possibly a few have been wrongly included, but it seems certain that this group is much the largest. Details of the age when the attacks started are given in Table III. Naturally in this group the patients were younger, and nineteen were under 20, fifteen from 20–29, and twelve from 30–39, leaving only seven over this age and only three over 50 ; 72 per cent. were between 15 and 39 years of age at the onset of their attacks.

The ætiology of the 42 with records of the paroxysms are shown in Table IV. The only points of interest are the rarity of ventricular attacks in the normal and rheumatic groups, and that just over half of the nodal attacks were found in the rheumatic group.

TABLE IV

TYPE OF PAROXYSMS IN DIFFERENT ÆTIOLOGICAL GROUPS

Type of Paroxysms	Ætiological Group				Total
	Normal	Rheumatic	Hyperpietic	Myocardial	
Supraventricular ..	16	13*	2	3	34
Ventricular	3	0	0	5	8
Number with cardiograms	19	13	2	8	42
Total number with paroxysms ..	59	19	8	14	100

* Of these 13, 7 were nodal.

CAUSATION OF PAROXYSMS

Although it is important from the practical point of view to decide whether there is organic heart disease or not, this does not explain the initiation of a

paroxysm. There must be some instability of the normal pacemaker, either absolute or relative to other parts of the heart muscle, and this may sometimes be shown by the occurrence of other abnormal rhythms. There was a respiratory sinus arrhythmia of sufficient magnitude to be unusual in 7 ; and extrasystoles were observed in 26, indicating that parts of the heart other than the S-A node were over-ready to initiate impulses. Two others showed nodal rhythm and sino-auricular block.

Peculiar changes were seen in the cardiogram of one patient. When lying on her right side there was a short P-R interval of 0·08 sec., a large wide R, and an S-T interval below the iso-electric level (Fig. 12). On her left side

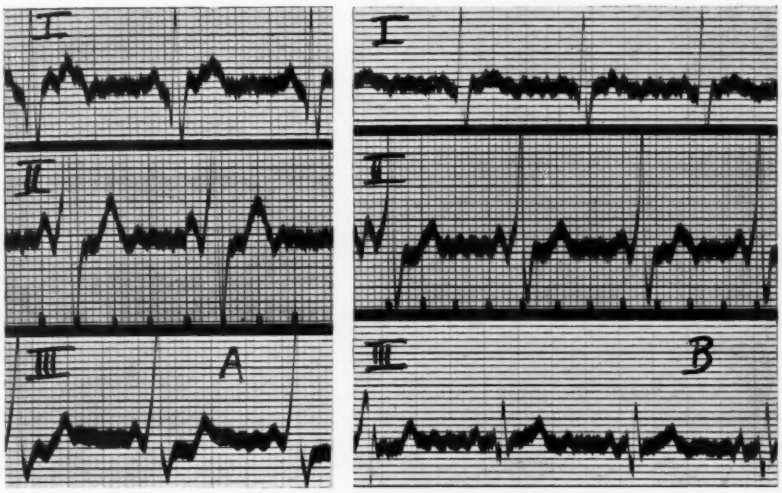

FIG. 12.—Changes which could generally be induced in one patient : (A) lying on the right side ; (B) lying on the left side (Case 30).

lead III changed, the P-R interval 0·14 sec., a smaller R, and larger S with upright T waves ; the QRS waves varied with respiration more in this position than on her right side. These two types were obtained frequently, and sometimes occurred apart from changes of position, suddenly from beat to beat (Fig. 13). On one occasion the type with a large wide R was obtained by holding her

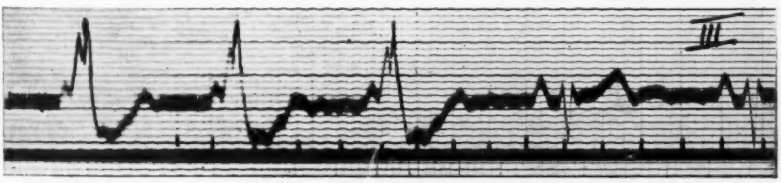

FIG. 13.—Spontaneous changes, illustrating the short P-R interval with complexes of bundle branch block type (Case 30).

breath after a deep inspiration, the S type being present at the beginning and again after expiration.

Wolff, Parkinson, and White (1930) published 11 cases prone to paroxysms with somewhat similar changes, for in the free intervals there was a short P-R interval and a bundle branch lesion, both being abolished by exercise. Evidently the S-A node is in some cases unduly over susceptible to outside influences, such as respiration or changes of position; and in others the relative excitability of the S-A node and the extranodal tissues is altered, extrasystoles resulting.

In addition to this underlying instability, some exciting factor must initiate the paroxysms from an ectopic focus. Many attacks, as is well known, start for no apparent reason and without any warning, but sometimes the patient knows the usual exciting factor, and more rarely may be able to initiate an attack voluntarily.

The beginning or end of a paroxysm are not often recorded except in those studied intensively in hospital, but we were fortunate in recording them in six patients. An unusual type where the onset and offset of short attacks were constantly obtained in many records is shown in Fig. 6 ; at one time it was hard to obtain a record without these changes, but now seven years later they are less frequent. The end of two attacks and another beginning soon after are shown in Fig. 14 ; deep inspiration seemed to cause the short returns of

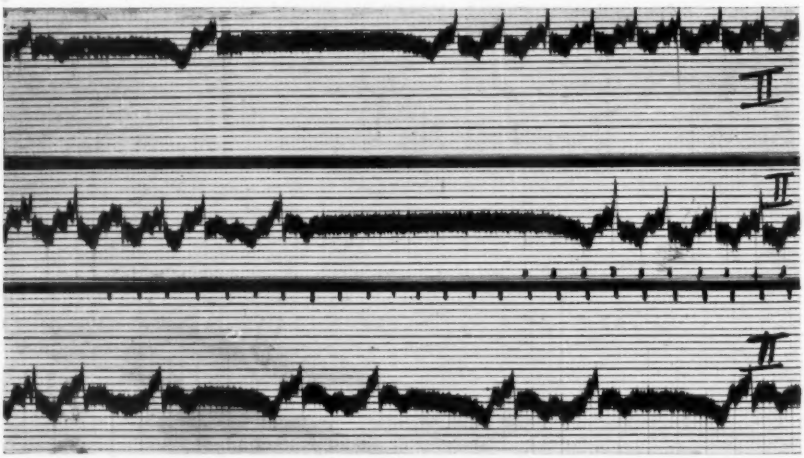

FIG. 14.—Onset and offset of paroxysms, rates 180 and 190, induced by deep breathing; lead II only (Case 82).

normal rhythm. Generally her attacks lasted for many hours, but that morning her condition was specially unstable. Short paroxysms from two other patients are shown in Fig. 15 ; the first, probably nodal, occurred spontaneously; the second could be produced almost regularly by deep inspiration. Another patient had her first attack when her doctor, examining her lungs after pneumonia, asked her to take a deep breath. The beginning of a paroxysm and a

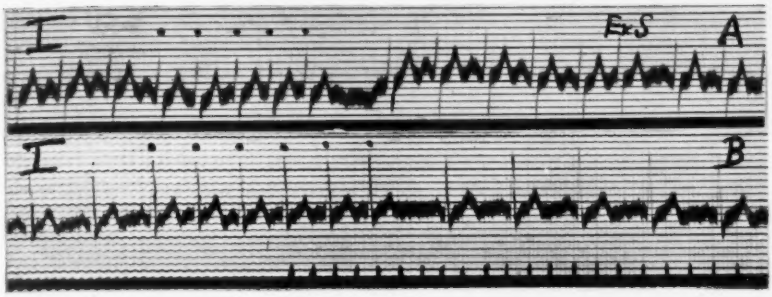

FIG. 15.—(A) Short nodal paroxysm, rate 174, occurring spontaneously in a woman with mitral stenosis (Case 46); (B) Short paroxysm, rate 158, induced by deep inspiration in a woman whose heart was normal (Lead I only).

complete short one are shown in Fig. 16 ; here again the short attack was exceptional, and there was no obvious exciting cause.

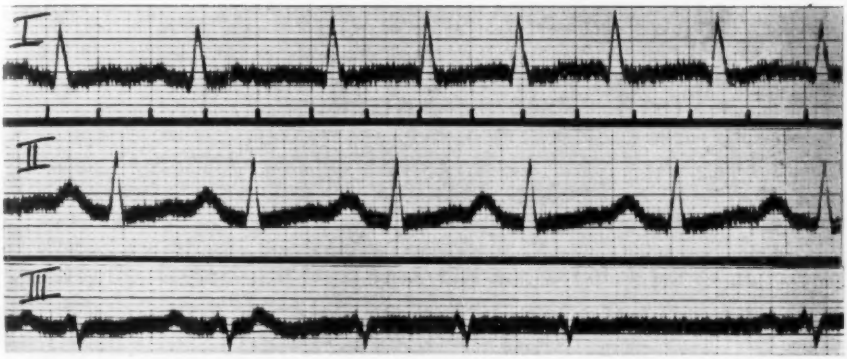

FIG. 16.—Onset of paroxysm, rate 171, in lead I, and complete short paroxysm in lead III ; occurring spontaneously in a woman with rheumatic heart disease (Case 60).

A paroxysm interrupting auricular fibrillation is shown in Fig. 17. This was the only time we were able to induce an attack by means of the stimulus

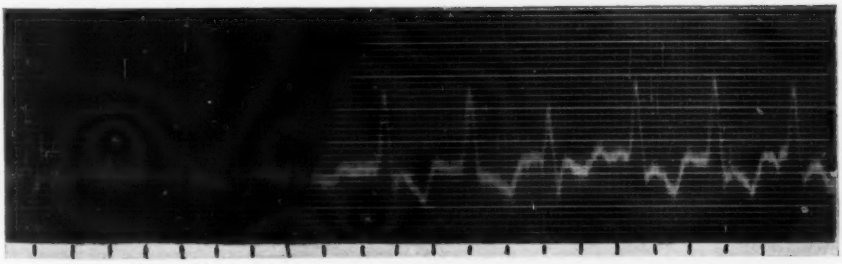

FIG. 17.—Paroxysm at a rate of 141, induced by a loud noise; in a man with established auricular fibrillation and myocardial disease (Case 56). In a long paper tracing the third beat of the paroxysm (shown above the word 'noise') was the only one that differed from the others.

which the patient thought was generally responsible for his attacks—in this instance the unexpected noise of dropping tins on a concrete floor. Another patient attributed several attacks to sudden unexpected noises, such as banging a door.

Sudden movements, such as stooping, turning on one side, jumping, or running upstairs, often start an attack, and where there is an ascertainable cause it is most often of this type. Such movements nearly always provided the stimulus in nine of our cases, for the most part with normal hearts ; but similar movements stopped attacks in six others, and this may be useful in advising treatment. One patient was particularly liable to an attack when playing tennis, especially at the moment he raised his arm to serve ; he was able to play if he took quinidine before a game. Another had his first attack when he leant out of bed to reach a urinal, and his most recent when he dropped his newspaper in the train and reached down to pick it up.

Such stories are far too common to be due to chance, though often no cause can be detected. Apart from these movements there is a somewhat heterogeneous group of conditions which seem to increase the instability of the S-A node so that any minor cause may excite an attack. Such conditions are anxiety, overwork, over-exertion, pregnancy, obesity, indigestion, smoking, and infection, and these will be considered shortly.

Anxiety.—The patient's state of mind is important. Emotional upsets, excitement, worry due to financial or domestic causes, or anxiety about the supposed gravity of the cardiac state may induce more frequent attacks ; in one the first and only attack occurred whilst he was engrossed in a game of chess, and this often happened in another patient. In a bank clerk the heavy work during the period of the War Loan conversion changed infrequent paroxysms into daily events. In many patients the attacks became less frequent and less important when they knew that the heart was otherwise normal. We are speaking of the effect of anxiety on normal people ; there is no special association with pathological anxiety states.

Infection.—There is little evidence that infection, acute or chronic, plays any important part. In two patients the first attacks started during convalescence from pneumonia and continued for many years. This might suggest a focus of myocardial damage, but there were no other signs of heart disease, and no such history of infections could be obtained in others. First attacks followed influenza, acute frontal sinusitis, rheumatic fever, tonsillectomy, etc., but six such cases in a hundred is not very convincing.

Chronic focal infections can be accepted as the cause of paroxysms only if their effective treatment produces an improvement. In this series results have been disappointing and no case has shown much improvement. At present there seems no real evidence for an infective group of cases. On general principles focal sepsis should be treated when it is present, but one should never assume that this will cure paroxysmal tachycardia.

Smoking.—There was some improvement in three cases when cigarettes were reduced. Reduction of smoking even down to complete abstinence failed to influence the frequency or severity of attacks in others. There are

people who are unable to smoke because of the frequent extrasystoles induced, and paroxysms of tachycardia occur in some of them ; but tobacco is not often the cause of paroxysms though over-indulgence may increase their frequency.

Exercise.—In six patients attacks were increased by exertion greater than that to which they were accustomed, e.g. working overtime, cross country running, and football when out of training ; and reduction of exercise led to improvement. In two others the first attacks had followed on exercise, in one a game of hockey and in the other long distance running, but they continued without such causes. In two exertion had the opposite effect ; one of them could generally stop an attack by walking or running ; the other, who was leading a sedentary life, was improved by daily exercise. Most of these had no heart disease, but especially in those with rheumatic hearts more frequent paroxysms were often due to their doing more than was advisable for the condition of their hearts.

Indigestion.—No organic disease of the stomach or intestine was found in any of these patients. In two, attacks usually occurred at a fixed time after a meal, and in three a period of indigestion led to an increase in attacks. In two others their frequency was greatly diminished by treatment of their indigestion, but two doctors were disappointed that effective treatment of their indigestion failed to reduce the attacks. Sometimes the disturbance of digestive function produced by the paroxysm is mistaken for its cause. One patient attributed his unusually long attack to indigestion, though he did not usually suffer in this way with shorter ones ; there was, however, congestion of the liver owing to a seven-day attack at a rapid rate. There were severe gastric symptoms, nausea, retching, and vomiting, in eight others. Spontaneous vomiting heralded the termination in three, and induced vomiting was the best method of ending an attack in others. Such symptoms were only observed in a minority, and flatulent dyspepsia is only rarely the cause. Nevertheless enquiries should always be made along these lines, and may reveal an effective method of treatment.

Allergic Conditions.—We have found no association between paroxysmal tachycardia and asthma, urticaria, migraine, or any conditions which are thought to be associated with allergy.

Pregnancy.—Five of these patients and several we have seen since were unfavourably influenced by pregnancy. In three the attacks were more frequent, in two they started at that time. On the other hand, two improved, one for the time and the other after as well as during her pregnancy. Anderson (1932) also has reported a case where paroxysms stopped during pregnancy and (1933) another where they occurred only at this time. In no case in our series except the one described (see p. 156) was there any reason to suppose that pregnancy should have been terminated on account of paroxysms. Meyer, Lackner, and Schochet (1930) reporting two cases stated they had only been able to find four or five examples of paroxysmal tachycardia in pregnancy ; we think they are by no means uncommon and are often mistaken for simple palpitation.

Obesity.—This is mentioned because in two cases, both comparatively young but over-stout, reduction in weight was followed by improvement.

Intrathoracic Disease.—This may cause paroxysms, and two patients, seen since this series, had their first attacks as an early symptom of a mediastinal neoplasm. In two others they occurred during the course of pulmonary tuberculosis. Graber (1925–26) has reported a case where lymphadenomatous glands were pressing on the vagus and we have recently seen an attack in a young man with a Riedel's goitre where the subsequent attacks of pain showed that there was pressure on the vagus. Paroxysmal auricular fibrillation is a more common sequel of thoracic neoplasms, especially when there is a pleural effusion.

SYMPTOMATOLOGY OF PAROXYSMS

Besides the sensation of palpitation complaint may be made of a variety of symptoms, any of which may be more or less prominent and exceptionally may mask the true nature of the paroxysm, especially in children. Rarely the patient may be unaware he is experiencing an attack, when, of course, the diagnosis depends on chance observation. The general symptoms, palpitation, weakness, faintness, dizziness, anxiety, or breathlessness, will not be considered in detail as they are of little special significance in themselves. Any degree of incapacity up to complete prostration may be met with, and may be attributed to any of these sensations. On the whole our experience corresponds with that of other observers—that the incapacity depends mainly on the state of the heart muscle, on the length and to a less extent on the rate of the attack, and on the mentality of the patient.

If the heart is healthy the symptoms are generally discomfort rather than real disability. One man thought it was sufficient if he came out of the scrum and took an easier position in the field when an attack started during a game of Rugby football. Another was able to carry on his routine work at the hospital and lecture, though he certainly found it more of an effort. At the other extreme some patients developed congestive failure. Many of those with rheumatic or other heart disease had little more disability than most of the normals, yet with severe heart disease any attack may be very grave. The importance of the duration and of the rate is discussed later. Shortly one can say that if an attack lasts for one or two days the symptoms and disability generally become important, even if the heart is healthy ; and that a healthy heart will support short attacks even at such a rapid rate as 200 surprisingly well.

Loss of Consciousness.—Unconsciousness during the attack is very rare. Two patients, both with heart disease, said that occasionally they had lost consciousness just after the onset. As the hundred cases represent several thousands of attacks, the rarity of loss of consciousness is obvious.

Anginal Pain.—In addition to discomfort or tightness in the chest, present in most attacks and of no special significance, twelve patients had more severe pain of an anginal type during attacks, some occasionally, others when the

the attack had lasted a definite number of hours. In ten it was felt beneath the sternum or just to one side and in two under the left breast ; sometimes it spread down the left arm (five times), down both arms (once), or up into the angle of the jaw (twice). Only in two was the pain really severe, and one of these had angina on exertion apart from his paroxysms. In four where nitrites were used the pain was eased. Seven of these twelve had hearts which appeared normal except for their attacks, and two had mitral stenosis only. The presence of pain, even of a severe type simulating angina, does not indicate an unfavourable prognosis ; this must be judged mainly by the condition of the heart during the free periods. Only one of these twelve has died, six years after the onset of the paroxysms and angina.

White and Camp (1931–32) have drawn attention to the occasional occurrence of anginal pain in paroxysmal tachycardia or fibrillation and the need for distinguishing it from angina of effort, but they do not stress how benign it may be under these conditions. The relationship of paroxysmal tachycardia to coronary thrombosis is discussed later (see p. 148).

Congestive Failure.—This was observed during the attack in nine patients. One, who so far as we could judge had no other evidence of heart disease, had experienced several attacks without failure, but was seen on the eighth day of a ten-day attack with commencing failure, when the rate was 196 and the blood pressure had fallen from 140 to 105. Two children with hearts that were otherwise normal developed failure in attacks lasting two or three days, but the rates were unusually fast. The remaining six had diseased hearts—four myocardial, one rheumatic, and one hyperpietic. In seven the failure became apparent on about the third day, in one with rheumatic heart disease within twenty-four hours. Not many attacks lasting three days were observed in normals, as they were less likely to seek medical advice, but the figures quoted indicate that the development of congestive failure is dependent as a rule on heart disease. Its occurrence does not seem so serious as might be expected, especially where the heart is normal or even well compensated in the free intervals.

The Rate in Paroxysms

The rate of one or more attacks was observed in 70 cases. It is stated by Lewis (1920) to lie between 160 and 200 usually, with a range from 110 to 220. These upper limits are not high enough, and it is not very rare for the rate to be up to 240 or even faster. In our cases the rate was between 160 and 200 in 45 per cent. and between 140 and 240 in 90 per cent.

In three the rate was less than 140, viz. 136, 126, and 124. The last, who had frequent nodal attacks, is illustrated in Fig. 7. At such a slow rate the diagnosis may be difficult, and here it was not made until she was seen in an attack.

In two children the rate was over 250 (272 and 300, see Fig. 1). They were exceptional in many ways and have been described elsewhere (Campbell, 1937) ; the second may have been paroxysmal flutter. In four others (all supra-ventricular), the recorded rates were 248, 230, 240, and 234 (see Figs. 8*b*

and 18). Two had no evidence of heart disease and had already had attacks for fifteen and seventeen years. The other two had rheumatic lesions.

TABLE V

HEART RATE IN PAROXYSMS

Type of Paroxysmal Tachycardia	Number of cases recorded at each Rate							Total
	120–139	140–159	160–179	180–199	200–219	220–239	Above 240	
Nodal	2	5	5	1	2	2	0	17
Other Supra-ventricular ..	1	6	6	5	3	3	3	27
Ventricular ..	0	1	2	3	3	1	1	11
Paroxysms without a cardiogram	0	3	8	8	6	3	2	30
Total	3	15	21	17	14	9	6	85

Table V summarizes the findings and shows how the rates were distributed among the different types of paroxysms. There were cardiograms of 55 attacks from 42 patients (two from the same patient being counted because the rate was sometimes different), and of 30 others observed clinically. Slow and fast rates were found in each type. Analysis in various ways shows few conclusions except that nodal attacks tend to be a little slower and ventricular attacks a little faster ; and that sometimes it is a bad sign when the rate of the attacks in any patient falls during the course of months or years.

In the nodal attacks the average rate was 170 and the commonest range was 140 to 180 ; one fallacy may be that at the slower rates it is easier to distinguish the P wave and so to diagnose nodal tachycardia. In the other supra-ventricular attacks the average rate was 187 and the usual range was 150–230 with more below 190. In the ventricular the average was 198, the usual range was the same, but more were above 190.

The presence or absence of disease did not appear to influence the rate, the average for the normal cases being 190, for the rheumatic 178, for the myocardial 180, and for the hyperpietic 185. The range in all the larger groups was much the same and included fast and slow attacks.

The rate in different attacks is said to keep about constant. This is our general experience and cardiograms obtained in different attacks from several cases were practically identical after an interval of years. In many patients, however, they varied by as much as 10 to 15 per cent. In short attacks the rate may vary considerably within a few minutes. Here it may represent a different ectopic focus.

Where rates were accurately observed in several paroxysms one third showed a difference of between twelve and thirty beats per minute ; most of these had heart disease. The period over which paroxysms have occurred did not in itself cause a change in rate. The changes were not more common

in any one type of attack or in different diseases of the heart. There was some evidence that as the efficiency of a diseased heart lessens, the rate of the paroxysms may fall ; but others observed up to the time of death maintained a constant rate to the end. For example, in two a fall of 12 and 30 beats per minute preceded the onset of fibrillation, and in two others the rate slowed as they became worse—26 and 22 beats per minute. An increase in rate as the myocardium fails is less common.

Comparing the rates recorded graphically with those counted personally, there is no reason to think that the " clinical " rates are not accurate, except that there were fewer under 160, perhaps because paroxysms at a slow rate are less likely to be diagnosed without a graphic record. The rate should always be taken for half a minute. Generally when the rate is said to be uncountable it is because an attempt has been made to count the pulse by the finger instead of the heart by the ear.

The Length of Attacks

Paroxysms last for hours much more commonly than for days. Though variable, they tend to have a more or less customary length in any one subject, who can generally report that the attacks last between, say, two and eight hours, or less than half an hour, or more than a day, and is surprised if his past experience does not prove a useful guide of what to expect. In two patients, for example, the length of many of their attacks were recorded over a series of years. In one (Case 30) there were 122 paroxysms in eleven years (1927–38) and the length of 89 of these known ; the number of paroxysms of different lengths was as follows :

1–4 hours	8
5–8 „	19
9–12 „	24
13–16 „	14
17–20 „	5
21–24 „	10
25–48 „	7
17 and 29 days	2

In the other (Case 70) the length of 88 paroxysms in the years 1924–32 was recorded as follows :

Less than ¼ hour	55
½–1½ hours	15
2–3 „	9
4–5 „	8
8 „	1

In these two, though there is variation round the customary length in each case, it is easy to give an average range and to say that what would have been a long attack for one would have been a short attack for the other.

More than half of these patients generally had paroxysms of less than two

hours, and in seven only did they habitually last for a day or more, even though those with longer attacks are more likely to come under observation. Table VI shows their usual duration in this series, though often it was difficult to decide such precise limits. The longer attacks occurred a little more often in those with heart disease, but the difference was not great.

TABLE VI

THE CUSTOMARY LENGTH OF PAROXYSMS IN 100 PATIENTS

The Customary Length of Paroxysms	Patients with no Heart Disease except the Paroxysms	Patients with Heart Disease	Total Number of Patients
Up to 30 minutes	23	12	35
„ 2 hours	15	11	26
„ 6 hours	12	6	18
„ 12 hours	5	5	10
„ 24 hours	1	3	4
Over 24 hours	3	4	7
Total	59	41	100

The longer paroxysms are of more importance, for the distress and disability and even the likelihood of congestive failure are increased. In addition to the 7 that habitually had attacks lasting more than twenty-four hours, 22 others had some of this length, making a total of 29 with some or all of their attacks lasting more than a day. The attacks were not often this length and never greatly exceeded it in ten of these, six with heart disease and four without, and did not produce serious symptoms, or any change in the general condition.

The remaining 19 with long attacks are illustrated in Table VII. There were six (including two small children) with hearts that were normal except for the paroxysms and four with rheumatic heart disease, most of the remainder falling into the myocardial group. The long attacks were least frequent in those without other evidence of heart disease—11 per cent. of the normal, 21 per cent. of the rheumatic, and 33 per cent. of the other groups having these long attacks.

The rate in these longer attacks was generally faster than the average for all the paroxysms ; in only three was it under 180 and in ten of the nineteen it was 200 or over, while among the other cases there were more under 180 than at 200 or over. The average rate in these long attacks was 208 (or 198 if the two exceptionally fast ones be excluded), instead of 186 for the series as a whole. All types of attacks were represented, but there was a much higher proportion of ventricular paroxysms than of the other types, nearly half the former lasting for more than twenty-four hours.

Even among these longer attacks relatively few lasted for more than two or three days, and they were of this length in eight patients. In two they lasted 4 days ; in two up to 7 days ; and in five, 10 days ; only in two did they last longer than 10 days. One of these (Case 15) is described in the

TABLE VII

PAROXYSMS OF UNUSUAL LENGTH

Case Number	Age	Ætiological Group	Paroxysms		Length of Paroxysms	
			Rate	Type *	Usual—in hours	Less Usual
Seven patients whose paroxysms generally lasted more than 24 hours						
5	21	Myocardial	210	V.	Three attacks only of 5 to 10 days.	
15	24	Myocardial	190	—	8–48	7–8 days, three 14 and 17 days and 4 weeks
69	1	Normal	272	V	48	Several days.
78	1	Normal	300	S.	Two attacks only of 3 days.	
84	27	Rheumatic	175	N.	6–48	Up to 7 days.
85	20	Rheumatic	200	S.	12–36	Up to 4 days.
88	40	Normal	—	—	10–30	Up to 3 days.
Twelve patients whose paroxysms generally lasted for a few hours only						
11	30	Myocardial	205	V.	1–6	Several of 10 days.
16	53	Myocardial	160	—	12–24	One of 7 days.
30	17	Normal	220	S.	10–18	Two 29 and 17 days ; several 36–50 hours.
36	44	Rheumatic	240	S.	¼–1	One of 2 days.
37	47	Syphilitic	—	—	2	One of 2 days.
43	48	Normal	190	S.	1–6	One of 10 days ; several 1–2 days.
55	41	Myocardial	200	—	4–12	Several of 3 days.
61	50	Hyperpietic	172 to 204	—	1–48	Several over 2 days.
68	25	Myocardial	200	—	Variable	Four attacks 7–10 days.
75	20	Normal	180	—	½–3	Sometimes up to 3 days.
80	41	Rheumatic	217	N.	½–1	Sometimes up to 4 days.
82	12	Normal	197	S.	2–12	Some up to 7 and 10 days.

* N, nodal ; S, supraventricular ; V, ventricular paroxysms.

section on prognosis ; an attack in which congestive failure developed lasted 17 days. There had been shorter ones for sixteen years, but longer attacks recurred and he died five years later in one that lasted more than 28 days. The other, aged 38, had suffered from attacks for twenty years and during five years' observation had about ten a year lasting generally for 10–18 hours and never more than 48 hours. She was admitted to another hospital when one attack had lasted 24 days and the heart rate remained over 200 in spite of treatment with digitalis. As it still continued after 29 days she was transferred to Guy's Hospital, but it stopped during her journey. She soon felt as well as usual, though for a few days the T waves were rather flatter. The frequency and duration of her paroxysms were not permanently increased (see Table IX), and in other respects her heart could be passed as normal. After another seven years she had a second long attack of 17 days treated with digitalis in her local hospital. A cardiogram of a subsequent attack showed no evidence of progressive myocardial disease as it was almost identical with the first of 1927 (Fig. 18), except for some left axis deviation.

These attacks of exceptional length are very unusual and naturally more serious, but it is possible for them to occur without there being any disease of

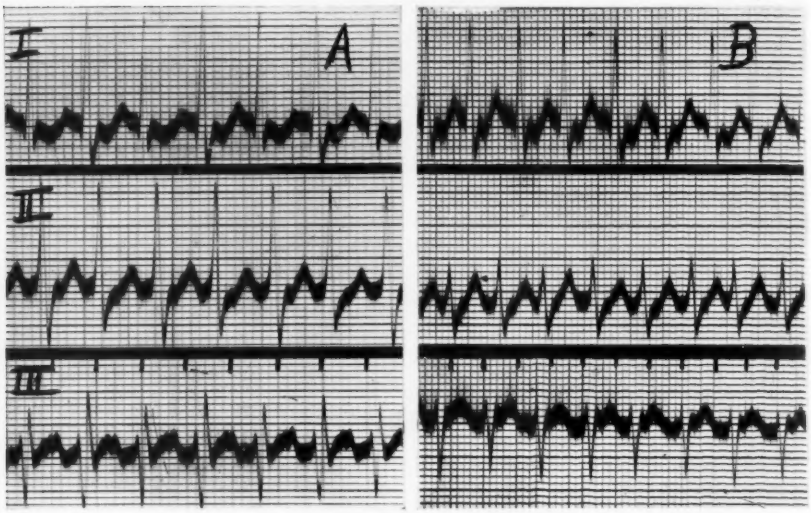

FIG. 18.—Two paroxysms from the same patient : (A) in 1927, rate 230 ; (B) in 1938, rate 207, (Case 30).

the heart and for complete recovery to take place. The great majority of paroxysms last for hours rather than for days, but 30 per cent. of our patients had one or more attacks lasting for a day, 10 per cent. of these very rarely, 10 per cent. occasionally, and 10 per cent. habitually. Only two had attacks lasting longer than ten days, though Gallavardin (1930) in a paper on paroxysmal tachycardia of long duration found that 11 out of 160 cases had some attacks as long as this.

TREATMENT

The treatment of the patient during a paroxysm and his management during the free intervals must be distinguished.

During an attack rest should generally be advised, but rest in bed is not needed unless special symptoms or the condition of the heart at other times make it advisable. Pressure on the vagus or on the carotid sinus is often effective, and also many tricks of sudden movement (e.g. bending down to open a low drawer) ; perhaps a forced expiration with a closed glottis after a deep inspiration is the most generally successful. If an attack persists towards evening, bed with a sedative or hypnotic will generally lead to its arrest.

Quinidine, 5 grains, repeated if necessary in two hours, is probably the most useful medicinal treatment. The cessation of attacks after digitalis has often been reported, but it is doubtful if it is effective, and generally it should not be given unless indicated by the state of the heart apart from the paroxysms or by the development of failure in a long attack. If there is any likelihood that the attack may be paroxysmal auricular flutter, full digitalization is indicated, both from the point of view of diagnosis and treatment.

Of newer remedies mecholin, 15 mg. intramuscularly, has often been successful in reported cases, but has been unpleasant and not very successful in our experience. So far prostigmin, 0·5 to 1·0 mg. subcutaneously, has seemed to us more effective and harmless to use ; but it is early to decide its special indications and limitations. Morphia is not desirable in ordinary recurrent attacks, but may be used and may be effective if an attack is causing serious anxiety to the medical attendant.

After the paroxysm the most important question is to decide the condition of the heart. If thorough examination justifies it, reassurance about the nature of the attacks is often all that is called for, and this alone will sometimes greatly diminish their incidence. The general regulation of the patients' life as regards food, work, and exercise must next be attended to, and in many cases a sedative such as bromides will be all else that is needed. If in spite of this the attacks are frequent and troublesome regular administration of quinidine for a time, 3 to 5 grains, t.i.d., will often succeed. Regular digitalization is rarely indicated and has not been useful in our experience. Several cases where these methods have been successful and some where they have not are given in this paper, especially in the section on course and prognosis.

COURSE AND PROGNOSIS

The Immediate Outlook at the time of a Paroxysm.

This is excellent, and however long a paroxysm has lasted, whatever the rate, and however ill the patient may seem, he may be expected to recover. The only exceptions are if the paroxysms are of the rarer ventricular type, or occur after the heart has begun to fail, or in the course of coronary thrombosis ; even so, though the risk is greater, he will generally recover from the immediate attack.

It is often stated that a patient may die in a paroxysm ; this is rare, and it creates just the impression it is necessary to avoid in the large majority, where there is no danger. Only five in this series of a hundred have died suddenly or during a paroxysm, including one from an overdose of morphia and one after a miscarriage ; and four of them had been liable to paroxysms for long periods (two for over 20 years and two for nearly 10 years) and had had hundreds of attacks before their deaths. Only one died soon after the onset of his paroxysms. Two at least, including the last, had ventricular paroxysmal tachycardia, and almost certainly died with ventricular fibrillation (Cases 5 and 79). A boy of seventeen under the care of Dr. Parkinson, who had suffered for five years from long and frequent ventricular paroxysms with gross congestive failure, provides more definite evidence. After an ordinary day with a short attack in the morning, he suddenly said that his heart had stopped and fell down dead, apparently after only a few breaths. From his words he must have known this was something quite different from the usual attacks and it was probably ventricular fibrillation. Because of this possibility the outlook in ventricular paroxysms is more serious, and this is one reason for trying to obtain an electrocardiogram of the paroxysm ; fortunately they are

much less common. With supraventricular paroxysms the outlook is excellent, apart from other unfavourable findings in the heart.

It is equally important when the attacks are only of recent onset to decide if they may indicate progressive heart disease. The age of the patient is some help, and of those whose first attack came after 40, only 25 per cent. had normal hearts ; while of those under 40, more than 60 per cent. had normal hearts. The following is an instance where paroxysms were the first symptom of commencing disease of the coronary artery. A man of 54 had a few paroxysms lasting two hours ; he had no pain and no cardiac symptoms previously. After a year he began to get angina of effort, and during the next two years this was induced more easily by diminishing amounts of exertion. There have been no further paroxysms. This is an important and not uncommon variety ; if paroxysms are of recent onset they are much more likely to be associated with organic heart disease in the elderly than in the young.

When a paroxysm occurs after coronary occlusion the immediate prognosis is not as bad as might appear, though it must add to the gravity. It generally stops spontaneously or after treatment, and the outlook depends more on the extent of the infarct. By chance none of these cases were examples of paroxysmal tachycardia secondary to coronary thrombosis, but one seen since illustrates the association. A man of 52 had a severe cardiac infarct, with pain for several hours in spite of morphia. His progress was satisfactory for ten days, until ventricular paroxysmal tachycardia started at a rate of 148. Quinidine, 30 grains daily, failed to arrest it, and he died after it had persisted for sixteen days. If, therefore, the patient is seen for the first time during a paroxysm, it is wiser not to make a final diagnosis of the heart condition at once. Murmurs may be difficult to hear, and if it has lasted long the blood pressure may have fallen and the heart may be dilated. The cardiogram may be abnormal for a few days after a long attack, so the complete examination should take place later than this. If at this stage the condition of the heart does not warrant a bad prognosis, the paroxysms may be looked on as an inconvenience rather than a serious matter.

The Ultimate Prognosis

This depends mainly on the condition of the heart in the free intervals, and to a lesser extent on the future frequency of the attacks and the effect, if any, they may have on the heart. Over 80 per cent. of these patients have already had attacks for more than five years and 47 per cent. for more than ten years, so that there has been ample opportunity to observe any unfavourable changes. The period which has elapsed since the onset of their paroxysms is shown in Table VIII. In the second and third columns they are divided into those who were alive when last heard of and those who have died, most of the deaths being due to the general course of their heart disease. In the last three columns they have been divided into three groups, those with no heart disease (including four with goitre), those with rheumatic heart disease, and those with hyperpietic

or myocardial disease ; as would be expected, the results are very different in these three groups.

TABLE VIII

LENGTH OF LIFE AFTER THE ONSET OF PAROXYSMS

Number of Years since onset of Paroxysms	Number of Cases			Number of Cases		
	Still living	Dead	Total	Normal group	Rheumatic group	Myocardial and Hyperpietic group
Up to 5 ..	16	2	18	13 —	3 —	2 (2)
5–9 ..	28	7	35	18 —	6 (2)	11 (5)
10–14 ..	13	3	16	10 —	2 (1)	4 (2)
15–19 ..	10	0	10	4 —	4 —	2 —
20–24 ..	4	1	5	1 —	2 —	2 (1)
25–29 ..	9	2	11	8 (2) *	2 —	1 —
30–39 ..	2	0	2	2 —	— —	— —
40–49 ..	0	0	0	0 —	— —	— —
50 and more	2	1	3	3 (1)	— —	— —
Total ..	84	16	100	59 (3)	19 (3)	22 (10)

* The numbers in brackets indicate those who have died.

1. *Normal Group.*—Where the heart was otherwise normal, serious complications were rare and a decrease in the frequency of the attacks from some change in the patient's life was quite as likely as an increase. The follow-up of these patients and close contact with many of them for years has convinced us that as a rule paroxysms do not cause progressive deterioration and still less disease of the heart, though some are very resistant to treatment and continue to have paroxysms with the same frequency over many years. Some examples may be given.

In one patient paroxysms started when he was 9 and continued through life. A few months before his death at 69, although he had developed lymphatic leukæmia which was held in check by deep X-ray therapy, he had no signs of heart disease, a normal electrocardiogram with good T waves, and was able to lead an active life (Case 1).

Another started having paroxysms when 50 ; at 75, though they were rather frequent and he had suffered from an enlarged prostate for seven years, he was still in business and did not notice any dyspnœa. There were no signs of cardiac or arterial disease, and his cardiogram was normal except for ventricular extrasystoles. He died three years later a few days after prostatectomy (Case 51).

A third was 68 when he was first seen and paroxysms had started at 23 ; they lasted up to two hours and he was rarely two weeks without. He was treated with bromides and luminal, and at 75, though his blood pressure was 180/110 and his heart a little enlarged, he had no complaint except of " a dulcimer constantly playing all the old tunes that had run through his life " ;

this had started two years before and was associated with increasing nerve deafness. His paroxysms were shorter and less severe (Case 66).

The cardiograms of these three cases are shown in Fig. 19.

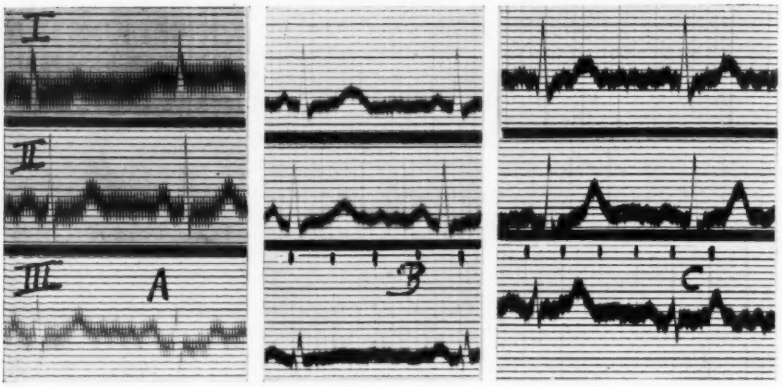

FIG. 19.—Normal electrocardiograms from three men who have had paroxysms for (A) 60 years (Case 1, aged 69); (B) 55 years (Case 66, aged 74); and (C) 28 years (Case 51, aged 75).

A fourth started having attacks when he was 7, and has rarely been three months without and sometimes has two or three in a month, lasting three to four hours or less commonly a day. He was seen when 56 with slight dyspnœa, with his aorta a little dilated and with broad QRS complexes in the cardiogram —findings which are not rare at his age. After another 10 years he has not aged much and there has been no substantial change in the condition of his heart or of his paroxysms, and he still leads an active life. He may die of heart disease, but in view of his long story this appears incidental (Case 89).

Table IX shows the frequency and length of paroxysms in a patient on

TABLE IX

FREQUENCY AND LENGTH OF PAROXYSMS IN CASE 30

Year	Number of Attacks	Length of Attack in Hours		
		Average	Usual Range	Total Hours
1927	6	13	10–16	81
1928	7	14	10–18	97
1929	10	17	13–24	173
1930	7	16	14–18	112
1931	16	20	12–24	320*
1932	7	21	8–48	146
1933	10	17	8–48	166
1934	19	9	4–12	164
1935	12	13	4–48	156
1936	11	9	6–12	103
1937	5	20	8–24	98
1938	4	25	12–36	76†

* Excluding attack of 29 days.

† Excluding attack of 17 days.

whom no treatment, medicinal or general, has had any influence. But after 29 years of paroxysms her health has not been impaired and during 12 years' observation there has been no progressive increase in their frequency.

All these were free from organic heart disease. Three others are still without any signs of heart disease or of impaired health after more than 20 years of attacks.

2. *Rheumatic Group.*—Here paroxysmal tachycardia might be expected to be more serious, but generally this was not so. Most of the rheumatic patients have been satisfactory (see Table VIII), and they have done as well as could have been expected had they been free from paroxysms. Details of three follow : the first was one of the few instances where her progress was downhill from about the time the paroxysms started ; the second was successfully treated with quinidine ; the third was not influenced by treatment, but got on well for eleven years with paroxysms and for another nine with fibrillation.

A woman of 35 with mitral stenosis had her first paroxysm while hop-picking, and subsequently short attacks occurred frequently up to three or four daily. Three years later fibrillation became established and she responded well to treatment with digitalis, but after another two years abandoned treatment for months and was admitted with congestive failure and a cerebral embolism, and died after three weeks. Autopsy showed extreme mitral stenosis, left intra-auricular thrombosis, and pulmonary infarcts (Case 46).

The second had rheumatic fever at 21 and paroxysms started at 33, after her third pregnancy. At 46, when first seen, the heart was slightly enlarged, with aortic incompetence. For 8 years most of her attacks were reported and their frequency and duration, shown in Table X, were greatly reduced with quinidine ; when this was stopped they became more frequent, after which she again resumed quinidine with success. Several attacks were observed, always at about the same rate, those recorded

TABLE X

FREQUENCY AND LENGTH OF PAROXYSMS IN CASE 70, SHOWING THE INFLUENCE OF QUINIDINE

Date	Period of Time in Months	Number of Paroxysms	Average per Month	Average Length in Hours (approx.)	Treatment
1924–25	10	39	3·9	1·5	—
1925–26	15	20	1·3	1·0	Quinidine, grs. 5.
1926	2	10	5·0	0·5	Pot. brom., grs. 45.
1927	4	6	1·5	0·5	Quinidine, grs. 5.
1927	2	20	10·0	0·5	Gentian and soda.
1927	7	16	2·3	1·5	Quinidine, grs. 5.
1928	12	13*	1·1	0·5	Quinidine, grs. 10.
1929	12	4†	0·3	2·0	,,
1930	12	6	0·5	3·0	,,
1931	12	4	0·3	1·5	,,
1932	12	3	0·2	1·5	,,
1933–35	36	15	0·4	—	,,
	122	85	0·7	1·5	With Quinidine.
	14	69	5·0	1·0	Without Quinidine.

* Including 6 in two weeks with influenza.
† Excluding several short attacks in two weeks with influenza.

clinically at about 240, those recorded graphically at 232, 225, and 233 (Fig. 8*b*). Her condition 14 years after she was first seen had not changed for the worse, and with ten grains of quinidine daily her attacks were still well controlled, about one each three months. Her blood pressure was almost the same as 14 years before and the heart seemed little larger. A paroxysm was recorded at a rate of 210, rather slower than before (Case 70).

The third had rheumatic fever when 5 years old, after which she was slightly short of breath. At 20 she had sudden palpitation when walking upstairs, and following this attacks lasting from one to twenty-four hours. She was first seen when 29 with a rather large heart and with mitral and aortic lesions. Paroxysms occurred frequently (Fig. 8*a*) and were not reduced in frequency by strophanthin, digitalis, or quinidine ; perhaps they were least frequent when she was taking bromides. Generally she had one or two attacks a week, and there was little change for two years. Fibrillation then became established when she was 31 ; the paroxysm before this lasting four days and seeming different because her heart had been irregular (paroxysmal fibrillation). Nine years later, after marriage and three years in India, fibrillation was still well controlled with digitalis (Case 52).

Paroxysmal Tachycardia and Auricular Fibrillation.—The relationship between these two may be considered here as it concerns rheumatic patients mainly. There is some connection, though it is not very close. Paroxysms interrupted fibrillation in one patient (Fig. 17). There were 7 others, 5 with rheumatic heart disease and 2 without, where fibrillation eventually became established and details are shown in Table XI. Another after having paroxysms

TABLE XI

ESTABLISHED AURICULAR FIBRILLATION FOLLOWING PAROXYSMAL TACHYCARDIA

Case Number	Heart Disease	Sex and Age at Onset of Paroxysms	Duration of Paroxysms before Auricular Fibrillation (in years)	Duration of Auricular Fibrillation (in years)	
				Patients who are dead	Patients who are alive
24	Mitral stenosis	F. 27	13	—	5
46	Mitral stenosis	F. 35	3	2	—
52	Mitral stenosis	F. 20	10	—	10
77	Aortic stenosis and incompetence	M. 40	28	—	1
80	Mitral stenosis	M. 42	8	—	1
38	Myocardial	M. 64	2	4	—
98	Normal	M. 57	6	—	3*

* Onset of fibrillation after an operation for pyonephrosis ; quinidine restored normal rhythm for a time only.

for twenty-four years began to have these interspaced with paroxysmal auricular fibrillation (both recorded) ; she still maintains normal rhythm after another three years (Case 70, Table X). A woman of 42 who had rheumatic fever when 21 sometimes changes at the end of her paroxysms to fibrillation. In one attack she was given quinidine and one and a half hours later her heart was irregular (Fig. 20) ; she said it was often like this towards the end (so it was not entirely due to the quinidine) and that if she kept still it would prob-

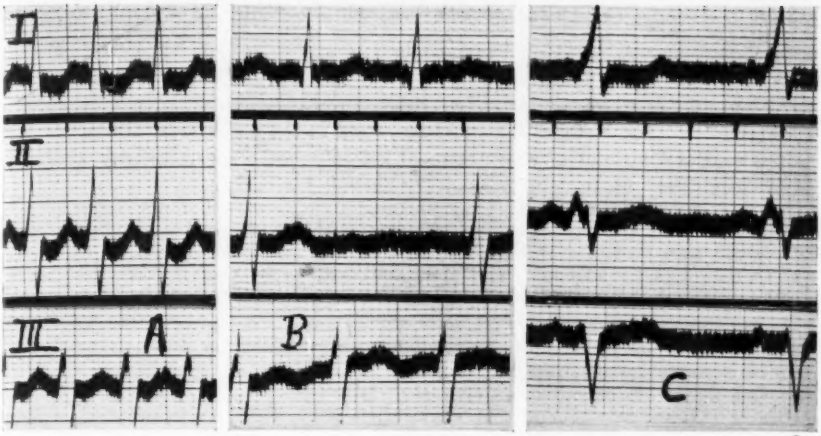

FIG. 20.—(A) Paroxysmal tachycardia, rate 214, from an obese woman with a rheumatic history; changing into (B) paroxysmal auricular fibrillation, rate about 103; and ten minutes later to (C) normal rhythm, rate 75.

ably stop. With great care she was moved to the galvanometer, which confirmed fibrillation, and in ten minutes normal rhythm returned (Fig. 20). Paroxysms of fibrillation in a rheumatic case often indicate that this rhythm will be established in the near future (Parkinson and Campbell, 1930); but this is not true of paroxysmal tachycardia.

3. *Hyperpietic and Myocardial Groups.* These patients have done less well than those with normal or rheumatic hearts, as nearly half of them have died. Two had syphilitic heart disease and eight had high blood pressure; in none of these did the occurrence of paroxysms seem of special significance, as the four who have died had serious heart disease which appeared to run its usual course. The other six in spite of their paroxysms have continued in moderate health—one after 26 years and another after 15 years, but possibly these two ought to be transferred to the normal group as the blood pressure may have developed later than and independently of the paroxysms. In all the prognosis was that to be expected from the condition of the heart quite apart from the paroxysms.

The myocardial group must be considered in rather more detail, and in view of the more serious outlook it is noteworthy that four of the eight with ventricular paroxysmal tachycardia belonged to this group. Four of the twelve in the myocardial group were elderly patients and would probably have come under observation for their heart condition quite apart from the paroxysms, which were unimportant compared with the other symptoms and findings; three of these have died.

The fifth was 58 and would not have been seen but for his paroxysms. Although he had no other symptoms his heart was enlarged and the cardiogram showed biphasic T waves in lead I and widened QRS complexes. Even so, although one paroxysm lasted nine days and led to congestive failure, they have not proved serious, since after six

years he is still well, with no progressive changes in the cardiogram or in the size of the heart (Case 16).

Three others would have passed as normal except for the cardiograms in the free intervals ; as all were between 26 and 40 the abnormality is the more remarkable. The changes that may be found in the cardiogram for hours or days after a long attack were not regarded as proof of heart disease, though they mean that this possibility has to be excluded with care.

One had a tuberculous hip excised when he was 11 and at 30 had his first paroxysm. Generally they lasted for an hour only, but on five occasions for seven to ten days so that he was in hospital. The abnormal cardiogram was the only evidence of heart disease, but now after 15 years he is becoming short of breath on exertion. He has some calcified areas in the mediastinum and at the apices of the lungs, so possibly some tuberculous disease spreading to the pericardium may be responsible for his paroxysms and for the abnormal cardiogram (Case 11).

One started having paroxysms when she was 12 years old. She was seen when 27 because although they were not frequent and only lasted five or six hours she felt ill for some days after them. Nothing was found except scoliosis and a cardiogram with flat T waves and rather wide QRS complexes. Five years later she was better, her attacks shorter, and her cardiogram more normal (Case 6).

The third has more frequent attacks which have ceased to trouble him since he found that they can be stopped easily by bending down to open a low drawer ; he has no other symptoms, but his cardiogram is still not normal (Case 68).

In the last two the abnormal signs in the cardiograms have become less, and now after six years they are in good health and without other symptoms. Possibly the paroxysms and abnormal cardiograms were the only signs of a temporary myocarditis from which there has been good recovery. Another patient, included as normal because nothing can now be detected in his heart (Case 43), had his first paroxysm when he leant out of bed during his convalescence from pneumonia ; he may have had a pneumococcal myocarditis and recovered completely except for the tendency to paroxysms, but there is no proof of this.

The remaining four (Cases 5, 15, 79, and 81) had abnormal cardiograms and some enlargement of the heart without obvious cause. Their ages when first seen were 21, 39, 37, and 18. They form an important sub-division as they include three of the five patients who have died suddenly or during attacks. All are fully described in the next section and in two the fatal result was probably due to ventricular fibrillation associated with ventricular paroxysms. The third is more disturbing because he provides the only instance where death was due to a paroxysm without the certainty of preceding heart disease, and where such changes as were found seem to have been caused by the paroxysms rather than to have caused them—a process which might be expected on *a priori* grounds, but is in our view most exceptional. Paroxysms had occurred for 21 years, and where so little could be found at the autopsy it is unlikely the enlargement of the heart was present at the start or that there could then have been any coronary disease ; the simplest explanation seems to be that attacks which were inconvenient rather than serious for 15 years began to last longer and to produce congestive failure, and that one lasted long

enough to cause his death. Without this case, we should have said more dogmatically that paroxysmal tachycardia alone never proved fatal. Even with myocardial disease, slight or serious, the prognosis is generally that of the heart disease and is not much affected by the paroxysms.

Patients who have Died

Some details about the 16 deaths during the period of observation (1925–38) will be given. At first sight this number suggests a bad prognosis, but in 4 the cause of death was nothing to do with the heart and in 7 others their length of life was quite up to the natural expectation of their heart disease, had there been no paroxysms. In 5 of the 16, however, death took place during a paroxysm and these must be considered fully, as even 5 out of 100 would be a serious proportion, were there no other reason for the deaths.

Two of the 4 patients whose deaths were not due to their hearts had suffered from paroxysms for 60 and 28 years and died from leukæmia and after prostatectomy respectively ; neither had any signs of heart disease even near the end of life. (Cases 1 and 51, see p. 149.) The third died two years after an operation for cancer of the breast with recurrences ; paroxysms are not uncommon when there are secondary deposits in the thorax, but here they occurred for some years before the neoplasm. The fourth died two years later from cerebral hæmorrhage.

In 7 patients death was caused by heart disease, but its course did not seem to be influenced by the paroxysms. One had a blood pressure of 200/110 and pulsus alternans ; a second, whose paroxysms began after fibrillation had been arrested by quinidine, died six years later with congestive failure ; a third had severe angina for six years before his death from coronary occlusion ; and a fourth had syphilitic aortic incompetence and an aneurysm. Two had rheumatic heart disease : one, with mitral stenosis and paroxysms for three years, died two years after the onset of fibrillation ; and the other, with a large heart and mitral and aortic lesions, lived eleven years after the onset of frequent paroxysms ; the seventh (Forman, 1931) had a calcified pericardium and died five years after the onset of his attacks.

So far as we know none of these 11 died unexpectedly or during paroxysms and we have no reason to think the length of life was shortened by them, though sometimes they were the first sign that the condition of the heart was starting to go downhill. This may be so, but is by no means the rule, and many patients with heart disease have done well without any progressive loss of reserve.

The remaining 5 are more significant, since they died suddenly or during paroxysms. The only one of these five that was thought to have a normal heart almost certainly died from an overdose of morphia. Of the other four two died suddenly and two with congestive failure.

The first was seen when his attacks, though infrequent, had already been present for 15 years ; and after another 11 years his wife wrote to report his death in a paroxysm. To further enquiries his doctor replied that as on previous occasions when

called in he had given half a grain of morphia, saying it was sufficient for the night ; but the patient had sent for a second doctor, who, unaware of the previous treatment, had given another injection of morphia, which probably caused his death (Case 28).

The second had mitral stenosis and moderate enlargement of the heart, and her supraventricular paroxysms began when she was 27 ; at 36 they had not greatly changed her condition and recurred about each six weeks and lasted for six to forty-eight hours (once seven days). She was admitted to hospital on the fourth day of an attack in the fourth month of pregnancy with anæmia and commencing failure, and as all efforts to stop it were unsuccessful her failure became worse. She lost a good deal of blood after a miscarriage and died on the seventeenth day. Here the attack was the immediate cause of the failure which led to her death, but the anæmia and the miscarriage were complicating factors (Case 84).

The third died suddenly when he was 45. He was refused for the army at 18 because of his heart, and again during the War at 26 ; he had some dyspnœa and palpitation, and when 37 lost consciousness after a paroxysm. Seen two years later, after a similar attack, he had a large heart mainly to the left, a loud systolic murmur, some widening of the aorta without high blood pressure, and a partial bundle branch block, and it was difficult to decide the exact nature of his heart disease. Several short paroxysms of ventricular tachycardia were observed at a rate of 186. Six years later we learnt that he had died suddenly while carrying an electric cooker downstairs. The coroner kindly supplied this report—" Heart 540 grams, hypertrophy and dilatation, chiefly of ventricles ; muscle thin and flabby with fibroid degeneration ; mitral and tricuspid valves themselves normal but incompetent ; coronary arteries moderate atheroma, orifices narrowed ; no rheumatic or syphilitic disease." Here it seemed probable that disease of the coronary arteries started at an early age and progressed till it led to his death. He was never an example of the common type, where the paroxysms were the main presenting symptom, because the physical signs and dyspnœa which led to his rejection from the army were present several years before the paroxysms (Case 79).

The fourth also died suddenly. His health had always been good except for diphtheria at 9, when he was in bed five months with paralysis of the soft palate and dilatation of his heart. Though some extrasystoles persisted he became free from symptoms, and when 19 took up heavy work, lifting crates that weighed 72 lb. ; he was able to carry on with this and with boxing and football without dyspnœa. When he was 21 he woke up in a paroxysm, which lasted ten days at a rate of about 200. After three weeks he felt fit and returned to work, but had shorter attacks of one and five days. When first seen five months later, his heart rate was 72 with frequent extrasystoles. The blood pressure was 120/85, and there were no physical signs except slight enlargement of the heart. His cardiogram showed poor T waves and ventricular extrasystoles of two types. He was admitted to hospital next day, with a ventricular paroxysm at a rate of 220 (Fig. 3) ; the blood pressure was under 100, and already there was some enlargement of the liver and slight œdema of the feet. Pressure on the carotid sinus and digitalis and strophanthin failed to arrest the attack, which stopped on the fifth day after twenty grains of quinidine, the rate falling gradually from 220 to 194 as a result of quinidine, and then suddenly to 80. The signs of congestive failure quickly disappeared and within two days he felt quite well, but the changes in the cardiogram (Fig. 21) led us to keep him in hospital for three weeks, by which time the inversion of T I had disappeared. Because of these changes and because the paroxysms were ventricular and quickly led to congestive failure, his doctor was given a guarded prognosis. He kept well for two weeks and perhaps unwisely took tinct. dig., min. xx daily. His doctor wrote that as he was starting for hospital for his next routine visit he suddenly fell down dead ; there was no autopsy (Case 5).

The fifth patient started attacks when he was 24 and for the next 15 years they recurred each six or nine months, lasting from a few hours up to one or even two days. They were merely an inconvenience and he continued work as an engine driver.

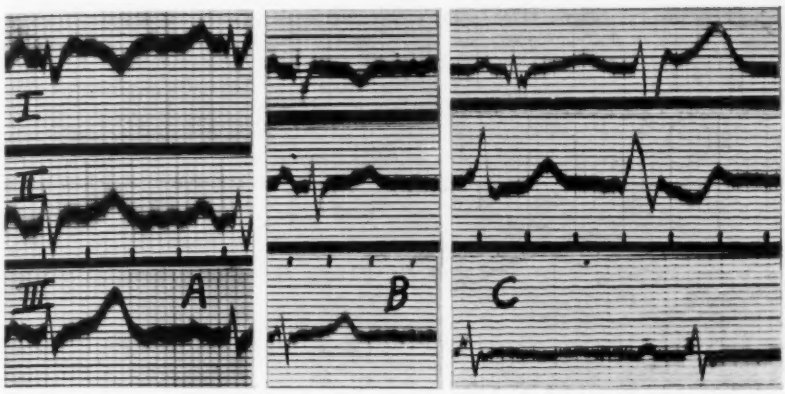

FIG. 21.—Changes simulating those of cardiac infarction, produced by a 10-day ventricular paroxysm ; (A) 2 days after, (B) 4 days after, and (C) 3 weeks after (Case 5).

In 1931, when he was 39, two attacks lasted 7 and 8 days, and for the first time he was ill enough to go to bed. Two months later he was admitted with congestive failure after 14 days of an attack which stopped when he had been given twenty minims of tincture of digitalis t.i.d. for three days. A cardiogram taken the next day when he seemed almost well showed some inversion of T in leads II and III, but ten days later these had gone, to be followed after a week by changes in the S-T intervals (Fig. 22).

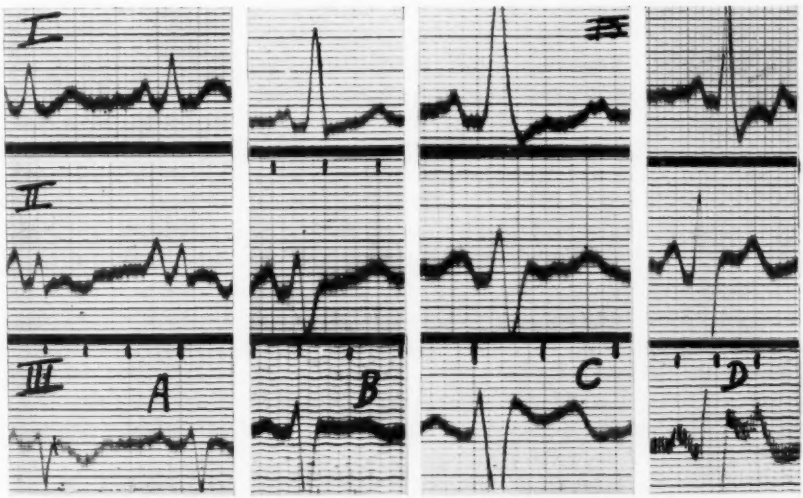

FIG. 22.—Changes after a 17-day paroxysm, simulating the changes after cardiac infarction ; (A) one day after, (B) eleven days after, (C) eighteen days after, and (D) four years after (Case 15).

Probably the inversion of T was due to the congestive failure and not to the digitalis or to coronary disease ; the findings at the autopsy described later and somewhat similar changes after other long paroxysms seem to justify this view.

Though he returned to work after some months he was never really well. In 1932 he had attacks of 8 and 14 days, and in 1933 nearly every month lasting about 4 days. In 1934 they generally lasted about 7 days and he was often away from work. Although there had been no more congestive failure he was able to do progressively less, and in 1935 had a pulmonary infarct during an attack. After this he gave up work and the attacks rarely lasted more than 4 or 5 hours. In 1936 his heart sounds were normal, his arteries soft, and his blood pressure 130/80 ; but an orthodiagram showed slight enlargement and the cardiogram (Fig. 22D) showed more depression of S-T in lead I than in 1933. Nothing else abnormal could be found ; he had no attacks in hospital and was advised to increase his activity slowly and to take quinidine regularly ; two months later he wrote that he had been better.

In 1937 he had another attack and after resting in bed three weeks he called in his doctor, but the paroxysm persisted, and there was progressive failure with jaundice and orthoponœa, On admission in the fifth week under the care of a colleague the heart rate was between 180 and 190, and he was dangerously ill. He was given 60 minims of tincture of digitalis, and during the night ¼ gr. morphia, but died next morning. At autopsy the heart weighed 450 grams (it seems unlikely that it was this weight the year before). There was no valvular disease, no signs of past syphilis, and very little atheroma of the aorta. The myocardium showed no scarring, and the coronary arteries were patent. There were 400 c.c. of slightly turbid fluid in the pericardium and the right pleural cavity contained a litre of blood-stained fluid. The lungs showed one very large and two smaller infarcts, as well as old scars. There was a large granular liver with nutmeg changes, and a firm dark spleen about twice the normal size (Case 15).

Summary and Conclusions

One hundred unselected cases of paroxysmal tachycardia have been studied and followed for some years. In 42 the diagnosis was confirmed by the electrocardiograph, in 30 by observation of an attack, and in 28 by the history alone. The criteria of diagnosis, when this has to be made on history, have been described ; the sudden onset of palpitation being the most reliable single symptom. There may sometimes be difficulty in distinguishing paroxysmal flutter and tachycardia.

Of the 42 attacks with graphic records, 8 were ventricular and 34 supraventricular ; 11 of the latter were nodal, but in many of the other 23 the site of origin could not be defined more precisely. Extrasystoles were observed between attacks in twenty-one, but were only of minor assistance in predicting the type of the attack. Ventricular paroxysms were very uncommon without serious heart disease.

There were 41 of these cases with heart disease, 19 rheumatic, 2 syphilitic, 8 hyperpietic, and 12 myocardial. There was no heart disease other than the arrhythmia in 59, though 4 of these had a goitre.

The rate was between 160 and 200 in nearly half ; it was between 140 and 240 in 90 per cent., but occasionally outside this wide range. There was no great difference between the various ætiological groups. Nodal attacks tended to be a little slower and ventricular attacks were rather more often above 190, but even above this rate ventricular attacks formed a small minority.

Paroxysms are generally of short duration, lasting for hours rather than for days. In 61 the customary duration was less than two hours, and in

another 28 twelve hours or less. There were 4 where it was about twenty-four hours and only 7 where it was longer than this. But 18 others, making 29 in all, sometimes had attacks lasting more than one day, viz. over twenty-four hours, 10 cases ; two or three days, 8 cases ; up to seven days, 4 cases ; up to ten days, 5 cases ; and two to four weeks, 2 cases. One third, therefore, of our patients had some attacks lasting more than a day, 10 per cent. rarely, 10 per cent. often, and 10 per cent. habitually. Long attacks included an undue proportion of the ventricular paroxysms and were more common in those with myocardial disease.

Paroxysmal tachycardia is a symptom rather than a disease. In a minority of patients it accompanies serious heart disease, when, of course, the prognosis is grave. Such cases are nearly always under observation for their heart disease before the onset of paroxysms. Ventricular paroxysms form a fairly large proportion of this group and are rare otherwise. In most patients paroxysmal tachycardia is not in itself of any grave significance. It is due to reflex causes more often than to any primary change in the heart muscle. This applies not only to the majority whose hearts are otherwise normal but also to most of those with rheumatic heart disease and to some of those with other myocardial disease.

There is no close association between paroxysmal tachycardia and paroxysmal auricular fibrillation. In some of the rheumatic cases and less often in others paroxysms of fibrillation may alternate with or replace paroxysms of tachycardia. In the rheumatic cases established fibrillation then becomes a possibility in the near future.

The prognosis of paroxysmal tachycardia as regards life is therefore excellent, unless it is of the rare ventricular type, unless appearing relatively late in life it is the first indication of disease of the coronary arteries, or unless before the paroxysms have started there is already serious heart disease. Three of these patients have lived fifty years after the onset of their paroxysms, another 18 for more than twenty years, and another 26, making 47 per cent., for more than ten years, and most of these are still in good health. Paroxysmal tachycardia does not produce heart disease, even when it continues throughout life, though one possible exception to this statement has been quoted. In general the prognosis depends on the condition of the heart muscle and should be decided without reference to the paroxysms. There is no constant tendency for the paroxysms to get worse as life advances, and usually some form of treatment can be found which will reduce the frequency and the discomfort produced by the attacks.

We wish to express our thanks to Drs. F. W. Gordon and S. S. Suzman for their help with parts of the later follow-ups.

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CHANGES IN THE CHEST LEAD ELECTROCARDIOGRAM IN CORONARY THROMBOSIS

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This investigation was undertaken in order to show the sequence of changes and the variations occurring in the fourth lead of the electrocardiogram in cases of coronary thrombosis giving the T_1 or T_3 type of tracing. Particular attention was paid to the earliest abnormalities to occur and the last to disappear. Sixty-four clinical examples of coronary thrombosis were studied in which electrocardiographic abnormality confirming the diagnosis was present. No cases in which the tracings were doubtful or unconfirmatory were included. Twenty-four were acute cases examined electrocardiographically within a few hours to four days of the attack (eight within twelve hours). The criteria of abnormality in the standard leads were RS-T changes and T wave inversion occurring in the acute group over a period of a few days or weeks.

The chest lead used was that known as lead IV F. In this the right arm electrode is attached to the left leg and the left arm electrode is in contact with the outer edge of the apex beat. A normal tracing consists of an upright P wave, an upward initial wave R of good amplitude, a downward wave S, and an upward T wave. The S-T segment is either iso-electric or slightly elevated. Significant abnormalities in this chest lead electrocardiogram are R waves of less than 3 mm., elevation of more than 2 mm., and depression of more than 1 mm. of the RS-T segment, and inverted, diphasic, or very high-voltage T waves. The evidence for the pathological significance of these abnormalities has been given in a recent paper by Bourne and Courtenay Evans (1938).

ANALYSIS OF CASES

The cases were divided into two groups, according to whether the tracing was of the T_1 or T_3 type. A small third group consists of two cases with changes only in lead IV, and a single case of an anomalous type is placed in group four. These groups were subdivided so as to analyse the early changes in the acute cases, and the later changes in all cases.

GROUP I. CHANGES IN LEAD IV F IN THE T_1 TYPE OF ELECTROCARDIOGRAM. This group comprises 40 cases, 14 of which were seen in the acute stage. *A. Early Stage.*—The initial deflection R_4 was absent in six, small in one, and

normal in seven of the 14 cases. Electrocardiograms were taken within eight hours of the thrombosis in three cases and the initial positive deflection was already absent ; in one of these a tracing showed an absent R wave two hours after the occlusion. In three cases, four lead electrocardiograms had been taken before the coronary thrombosis occurred, and gave a normal R wave ; after the thrombosis, in one case, a tracing taken on the fourth day showed an absent R wave ; the R wave was not altered in the other two cases either

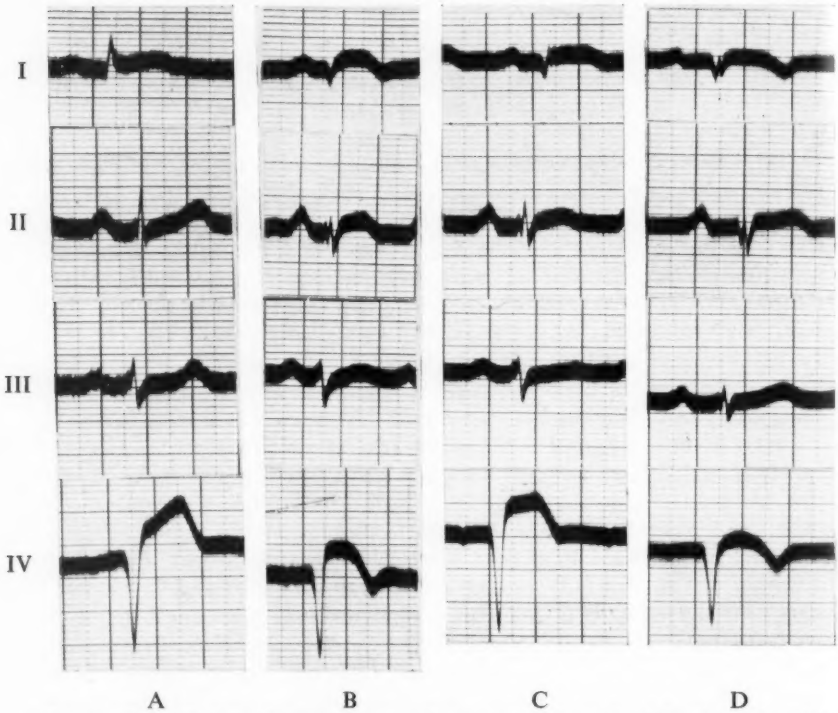

FIG. 1.—Serial T_1 type electrocardiograms from a case of anterior infarction, taken (A) 7 hours, (B) 4th day, (C) 5th day, (D) 11th day after coronary thrombosis. Note RS-T elevation and later inversion of T in leads I and IV ; monophasic negative initial deflection in lead IV (i.e. absent R_4).

in the acute stage or later. No significant changes have been noted in the S wave, but splintering was present once.

The RS-T segment was elevated in eleven cases, the average elevation being 3 mm. and the extremes 1 to 8 mm. It was depressed in three cases, the average depression being 1 mm. Humping with an upward convexity and slurring of the RS-T segment occurred in eight, but was absent in six. This humping showed any time after the fourth day, and sometimes remained for several weeks, but was not as a rule a permanent change.

The T wave was upright in eight cases, but in seven of them it became

inverted later. In the other six, the T wave was inverted in the first tracing taken after the thrombosis. Inversion of the T wave was never seen earlier than the third day, and appeared most commonly on the fourth day.

In limb leads, RS-T elevation occurred in seven cases in lead I. In three cases, RS-T elevation in lead I or in leads I and II was the only abnormal finding, when lead IV showed an absent R wave and gross RS-T elevation. In

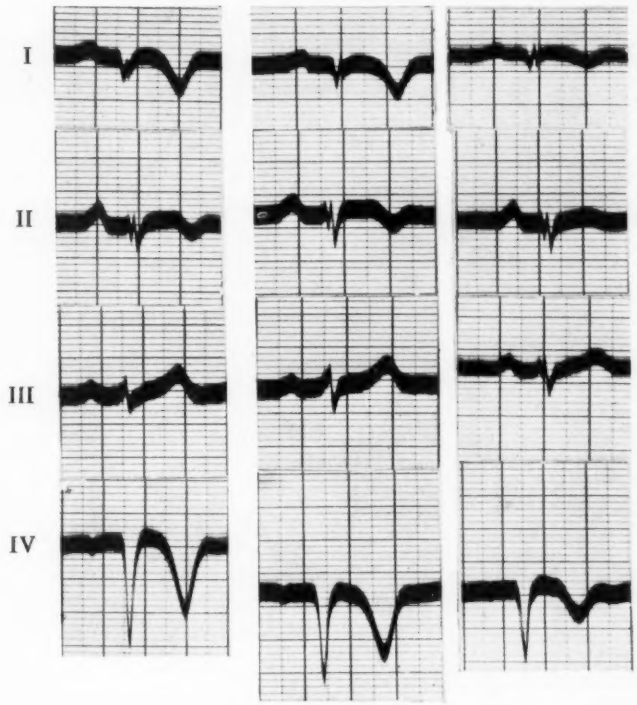

FIG. 2.—Later curves from same case as FIG. 1, taken (E) 21st day, (F) 32nd day, (G) 136th day after coronary thrombosis. Note disappearance of RS-T elevation ; persistence of T inversion in leads I and IV, and transitory T inversion in lead II ; absent R_4 persists.

one of these, T_1 and T_4 did not invert until the fourteenth day. T_4 tends to invert slightly earlier than T_1 ; in one case T_4 inverted on the fifth and T_1 on the twenty-third day.

B. Later Stage.—In lead IV, the R wave was absent in six of the fourteen acute cases ; three of these I was able to follow up and the R wave was still absent in two, 175 and 306 days after the thrombosis respectively. The other had a normal R wave after 137 days. Many tracings were taken at varying

intervals. Of the three others, two died in the acute stage and the third, still with an absent R, was lost sight of after 29 days.

Twenty-six other cases with the T_1 type of cardiogram were examined ten days to five years after the thrombosis. Twelve had an absent R wave, one an R deflection of 1 mm., and thirteen a normal deflection. Of the thirteen with an absent or very small R, one only showed recovery in later tracings, and in that case it followed cardio-omentopexy ; the remainder were unchanged.

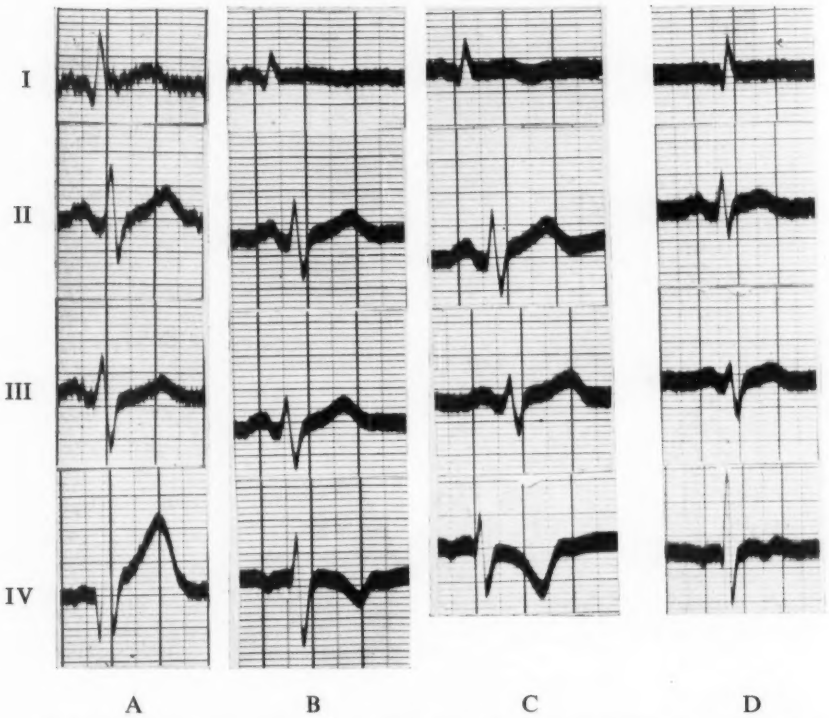

FIG. 3.—Serial T_1 type curves taken on (A) 1st day, (B) 5th day, (C) 29th day, and (D) 47th day after coronary thrombosis. Transient elevation of RS-T in leads I and IV ; inversion of T_4 precedes inversion of T_1. R_4 present throughout.

Most of these cases were repeatedly examined electrocardiographically for periods varying from five months to five years after the thrombosis.

Twenty-one of these forty cases with T_1 curves were followed up and seen on occasions five months to five years after coronary thrombosis. Analysis of lead IV showed notching of the QRS complex in three, W shaped QRS complexes in five, low-voltage QRS in one, and diphasic initial complex (QR) in three. The development of a diphasic initial complex (see Fig. 7 A) occurred a few months after the thrombosis in two cases, presenting at first an absent R wave and a large monophasic downward deflection. The S-T segment was

elevated in eight, iso-electric in eleven, and depressed in two. The T wave was inverted in twelve, diphasic in four, and normal in five. Three with diphasic T waves had formerly shown definite inversion. Of those with normal T waves, three were formerly inverted and return to normal occurred between the fourth and thirty-third week in one, between the eighth and twenty-fifth week in another, and between the fifteenth and thirtieth week in the other.

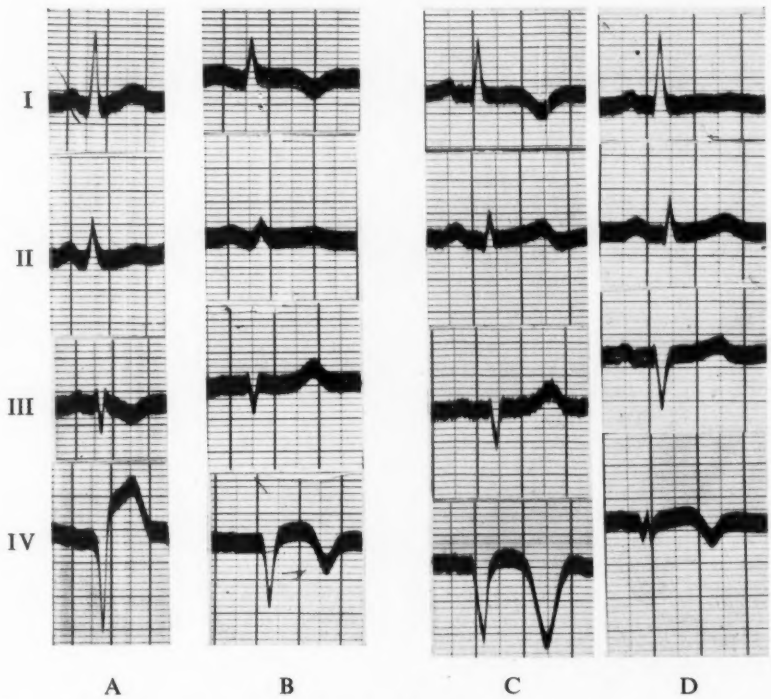

FIG. 4.—T_1 type curves in two cases of anterior infarction.

Case 1. (A) 6th day, showing earliest changes in lead IV only, i.e. Q-T elevation and absent R_4. (B) 20th day, showing inversion of T_1 and T_4 with upright T_3.
Case 2. (C) 39th day, showing inversion of T_1 and T_4, with absent R_4. (D) 291st day, T_1 inversion disappeared, but T_4 inversion persists.

In the limb leads T_1 remained inverted in nine, became diphasic in three, flat in four, and normal in five—inversion had been present in all. In the four cases which developed a flat T wave in lead I, T_4 remained abnormal ; and in the five which changed to a normal T_1, the T wave in lead IV remained abnormal in two.

GROUP II. CHANGES IN LEAD IV F IN THE T_3 TYPE OF ELECTROCARDIOGRAM.

A. Early Stage.—The initial wave R was normal in all of the eight cases ; the QRS complex was W shaped in one. The RS-T wave was depressed in six

(average 3 mm., maximum 7 mm.) and iso-electric in two. The T wave was upright in seven, and diphasic in one. The diphasic T wave was transitory, lasting a few days, and returning to normal on the fourth day.

In the limb leads, the RS-T segment in leads II and III was elevated in seven on the third or fourth day. Two cases were examined electrocardiographically a few hours after the attack, one showed immediate RS-T elevation

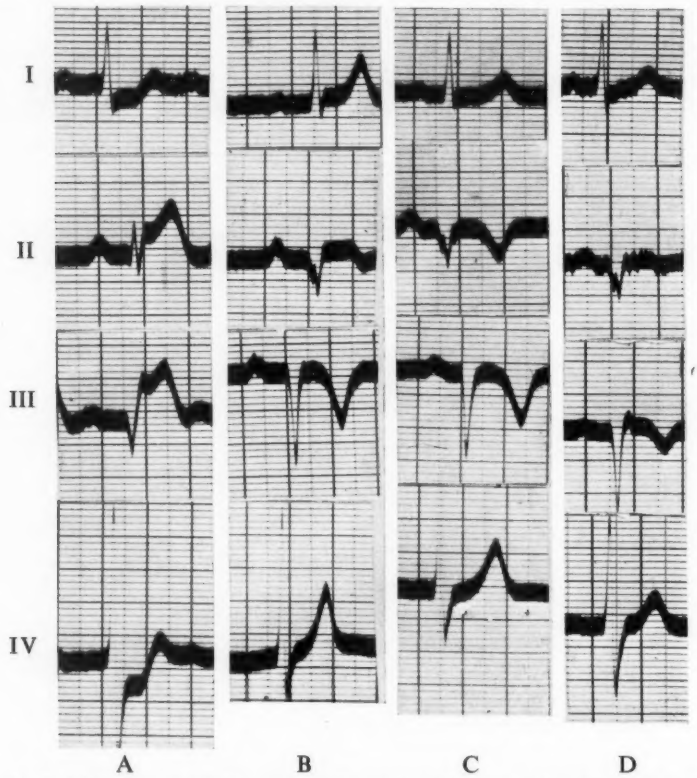

FIG. 5.—Serial T_3 type curves from a case of posterior infarction, taken (A) 4 hours, (B) 4th day, (C) 32nd day, (D) 172nd day after coronary thrombosis. Note elevation of RS-T in leads II and III and depression of R-T in leads I and IV, followed by inversion of T_2 and T_3.

in leads II and III ; the other remained iso-electric in the first tracing and gave the characteristic elevation on the fourth day. T wave inversion usually started on the third or fourth day, but one did not show inversion until the eighth. Six eventually had inversion of T_2 and T_3 ; two who died within the first seven days had no inversion. Post-mortem, one of these showed infarction of the posterior surface of the ventricles; there was no autopsy in the other. A Q wave was present in lead II in four cases, and there was an abnormally large Q in lead III in five cases.

B. Later Stage.—This is an analysis of eleven cases of coronary thrombosis with the T_3 type of electrocardiogram in which tracings were taken over four months or longer. In lead IV, the initial deflection R was normal in all cases. The RS-T segment was finally iso-electric in six, depressed in four, and elevated in one (formerly depressed). The T wave was finally upright in all eleven, and

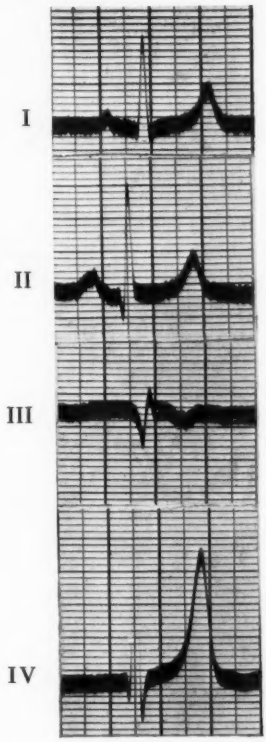

FIG. 6.—T_3 type curve 10 months after coronary thrombosis, showing Q_3 with slight inversion of T_3. Note abnormally large T_4 (18 mm.).

in only one was there ever an inversion (14th day). The T wave became of large amplitude in two (19 mm.), returning to normal size in one. Humping of the RS-T segment with a downward convexity was seen in one case only. In limb leads, the Q wave in lead III remained large in six. T_3 remained inverted in seven and T_2 in three cases.

GROUP III. EVIDENCE OF CORONARY THROMBOSIS IN LEAD IV ONLY.

A. Early Stage.—In two cases, evidence of a recent coronary thrombosis was seen only in lead IV, there being no changes in the limb leads. The electrocardiogram showed T_4 inversion with subsequent return to normal in one, and

T_4 inversion with humping of the RS-T complex in the other. The clinical evidence of coronary thrombosis was very strong.

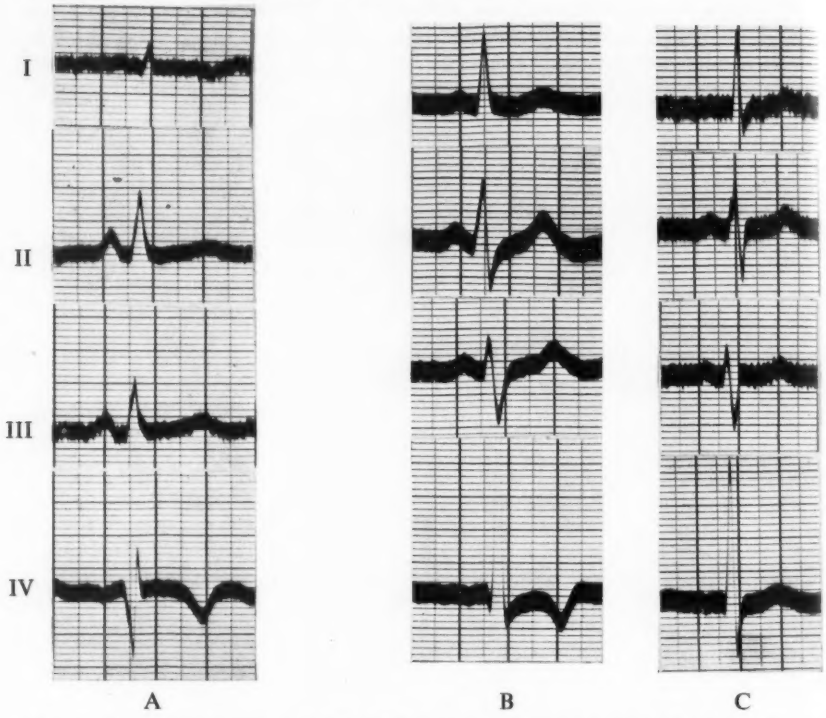

FIG. 7.—(A) T_1 type curve, 157 days after coronary thrombosis, showing diphasic initial complex (QR) in lead IV (previous curves showed monophasic negative initial complex). Curves taken on (B) 25th and (C) 49th day after coronary thrombosis, showing transitory inversion of T_4 and humped R-T_4 as only change.

B. Later Stage.—Four other cases, examined some time after the thrombosis, gave the only changes in lead IV, three by T wave inversion and one by an abnormally small R wave.

GROUP IV. ANOMALOUS CURVES AFTER CORONARY THROMBOSIS.

One acute case, which came to autopsy, showed elevation of RS-T_4, absent R_4, elevation of RS-T_1, and later diphasic T_2 and T_3. Auricular fibrillation, auricular flutter, and normal rhythm were present at different times. Post-mortem, a large infarct of the anterior and posterior surfaces of the ventricles.

POST-MORTEM EVIDENCE AND THE CHEST LEAD ELECTROCARDIOGRAM

Wood and Wolferth (1933) found, in experimental infarction in dogs, that depression of the RS-T segment occurred in anterior and elevation in posterior lesions. Using the standard lead IV, as in our cases, these findings would be reversed. Wood, Bellet, MacMillan, and Wolferth (1933) described thirty-one cases of acute coronary thrombosis, nineteen of which had anterior and twelve posterior infarction. The former had changes in the initial wave and in T_4 ; confirmation of the site of infarction was made by autopsy. Wood and Wolferth (1934), writing about abnormally large T waves in præcordial leads, describe such a case where autopsy showed an infarct on the anterior surface of the left ventricle. Willcox and Lovibond (1937) describe lead IV in six cases verified by autopsy. In three with posterior infarction, the correct diagnosis had been made from the limb electrocardiograms which were of the T_3 type. Of these, two had a normal lead IV, and the other an absent initial R, with slight elevation of S-T_4, but the post-mortem showed also patchy fibrosis of the anterior surface of the left ventricle. Of the other three, one had an apical infarction and the electrocardiogram showed a large T_4 ; another had an anterior and apical infarct and the tracing gave a T_1 type of curve with an abnormal R_4 and inverted T_4 ; the last case revealed an antero-posterior infarct and the tracing showed depression of R-T in leads I and II, elevation in leads III, and IV, and an absent R_4. Master, Dack, Kalter, and Jaffe (1937) describe the cardiograms of fifty cases examined post-mortem. Fifteen of these had anterior infarction with an absent initial R_4 in eleven, small R_4 in two, and normal R_4 in two. Thirteen had posterior infarction with a normal R wave in nine, small in four, and absent in one ; the latter also had an old partial occlusion of the left anterior descending coronary branch. In nineteen cases anterior and posterior infarctions were present ; the R wave was absent in four, and small in one-half of the cases. Two cases had infarction of the lateral wall of the left ventricle and one case infarction of the septum ; the QRS complex was normal in all three. Master only referred to the initial wave changes, not to the T waves.

In our series there were two autopsies. One of these showed recent infarction of the posterior surface of the left ventricle with sclerosis of the descending branch of the right coronary artery, but no arterial thrombosis. The tracing taken three days before death, on the fifth day, had elevation of the R-T segment in leads II and III, slight enlargement of Q_3, and humping of S-T in lead III. Lead IV was normal. The other case had extensive recent infarction of the whole of the left aspect of the interventricular septum and of adjacent parts of the anterior and posterior surfaces of the left ventricle ; the right coronary artery showed patchy areas of atheroma, and the left coronary at the commencement of the anterior descending branch was filled by adherent ante-mortem clot. The electrocardiogram as already described (Group IV) was anomalous.

Left Lateral Infarction.—Wood, Wolferth, and Bellet (1938) have described a type of electrocardiogram which they state is characteristic of left lateral

infarction. They produce post-mortem evidence and suggest that usually the circumflex branch of the left-coronary artery is occluded. The characteristics of the tracing are depression of RS-T in leads I, II, and IV, with a diphasic or inverted T wave in lead I, and an inverted T wave in lead IV. Auricular fibrillation is a common association. Electrocardiographic features may subside very rapidly, and after healing of the infarct all the changes may disappear from the tracing.

Three of my acute cases from Group I, with a T_1 type of electrocardiogram, had depression of RS-T_4, maximum 3 mm., and in two the RS-T segment in lead I was slightly depressed also. The R wave was present in all three. These are possible examples of left lateral infarction.

Summary and Conclusions

In the T_1 type of electrocardiogram after coronary thrombosis, the changes in lead IV occur in the following sequence. The initial wave R disappears immediately in half the cases, and elevation of the R-T segment, 3 mm. above the iso-electric level, occurs at the same time in three quarters of the cases. The T wave becomes inverted on the third or fourth day, synchronous with or slightly earlier than inversion of T_1, and sometimes many days earlier. At the same, time, the R-T elevation diminishes and, in half the cases, changes any time after the fourth day into a humped upward convexity with broad slurring of the curved portion. Return of RS-T to normal, with disappearance of the humping, occurs after several weeks. T wave inversion disappears in half the cases between the third and sixth month, but in behalf the cases it persists. The initial wave R, when once absent, usually tends to remain absent ; the initial complex in these cases sometimes becomes diphasic (QR).

In the T_3 type of electrocardiogram, RS-T is depressed 3 mm. below the iso-electric level, returning to normal within a few days in two thirds of the cases. The initial R wave is present, the S wave is often small, and sometimes a W shaped complex is seen. Later, humping of the S-T segment with a downward convexity occurs in a few cases, but is not common. The T wave is upright and tends to increase in amplitude ; it may attain 13–19 mm. in the second or third weeks. As a rule the changes in lead IV, after the initial RS-T depression has passed, are slight and not very helpful in diagnosis.

Post-mortem evidence suggests that T_1 and T_4 inversion, and absent R_4 with elevation of RS-T in leads I, II, and IV indicate an anterior infarction, and that T_2 and T_3 inversion with depression of R–T_4 indicate a posterior infarct. Where absence of R_4 and T_4 inversion are associated with inversion of T_2 and T_3, the evidence points to infarction of both anterior and posterior ventricular walls. The third type of occlusion, involving the left circumflex artery and causing left lateral infarction, shows RS-T depression in leads I, II, and IV, with inversion of T in leads I, II, and IV. These changes are often of a transitory nature and may be associated with auricular fibrillation.

I am indebted to Dr. Geoffrey Bourne for help and advice in carrying out this work, and I wish to acknowledge a personal grant from the Medical Research Council.

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TRAUMATIC ANEURYSM OF THE LEFT VENTRICLE

BY

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From St. Vincent's Hospital, Dublin

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Although aneurysm of the heart is still thought to be a somewhat rare and unusual pathological condition, recent work shows that it occurs not infrequently after coronary occlusion and infarction. Parkinson, Bedford, and Thompson (1938) estimate that it occurs in 9 per cent. of cases of cardiac infarction examined at necropsy. In the absence of coronary disease, aneurysm of the heart appears to be exceedingly rare ; and trauma as an exciting cause is seldom referred to in papers on the subject. Warburg (1938) in a recent monograph has collected, from papers and reports dating back to the year 1676, 197 cases of cardiac lesions due to non-penetrating injuries, but of these only two or three appear to be instances of traumatic aneurysm of the heart.

The case recorded here is one of traumatic aneurysm of the left ventricle, due to a non-penetrating injury to the thorax. Although the diagnosis was not made during life, the case presents some interesting points clinically and medico-legally.

He was a man, aged 59 years, a non-smoker and life abstainer. While driving a motor car he was involved in a head-on collision with another car on July 12, 1936. Immediately after the accident he noticed pain in his knees only. He was able to help some other people who had been injured, and later with assistance he changed the wheel of his car. Following this he experienced sudden severe pain in the region of his heart and became short of breath. An injection of morphia was given, and as a condition of shock still continued, he was taken by car to a neighbouring town. On arrival there he was seen by a doctor, and next day was admitted to the local hospital, as the præcordial pain had again become severe and was accompanied by marked dyspnœa. A tentative diagnosis of pericarditis was made. While in hospital he developed a left-sided pleurisy, and an X-ray photograph is said to have revealed a fracture of one of the left lower ribs near the vertebral junction. He was discharged from hospital on July 23 and returned to his own home by train.

On July 30 he was seen by a surgeon, who reported as follows : " There were tender points over the left costal cartilages and over the xiphisternum, and

over the eleventh rib at the back. The patient showed abnormal nervousness and increased reflexes. He breathed with short gasps and could not take a deep breath ; his pulse rate was 98. In my opinion the patient has suffered a crushing injury to the chest, probably due to striking against the steering wheel at the moment of collision. The crush has injured the costo-condral and costo-sternal junctions over the fifth to seventh ribs, which interfere with normal breathing. He will be confined to his house for at least a month, but may be able to do some light work in three months' time. His expectation of life has been adversely affected."

On August 6 he was seen by the present writer on account of an acute heart attack on the previous evening. On examination he was in great distress from severe shortness of breath, with a feeling of constriction and pain in the chest. The heart was rapid and irregular (auricular fibrillation). The blood pressure was slightly raised and there was distinct præcordial tenderness. On careful inquiry there was no history of any significant illness previous to the accident and nothing to suggest that there ever had been any symptoms of cardiac disability. With complete rest, sedatives, and digitalis the cardiac condition seemed to undergo improvement. As the heart rate became steadier the sounds became of a " tic tac " quality, and the breathing, though less embarrassed, always seemed to occur in short, sharp " whiffs." From time to time he experienced attacks of pain like angina of varying severity, and morphia had to be given on several occasions. Throughout his illness he was nervous and apprehensive, and there was always some slight tenderness over the præcordium.

At the end of August he was allowed up for a short time, but as this caused respiratory distress he was ordered back to bed. After a further period of rest in bed about five weeks he was again allowed up and arrangements were made for electrocardiographic and X-ray examinations. Unfortunately these investigations were not carried out, as he developed a left-sided pleural effusion early in October. On October 18 he died suddenly in an acute heart attack.

Post-mortem Examination (Prof. J. McGrath).—The body was that of a well-built man, of healthy appearance, with a moderate amount of fat. There was no evidence of bruising on the front of the chest. On opening the chest, the left pleural cavity was found to be almost completely filled with a large quantity of clear, straw-coloured fluid. The lower lobe of the left lung was entirely collapsed, as was also the lower portion of the upper lobe. The left lung itself, except for the above findings, appeared normal. There was the usual emphysema for a man of his age. The right lung showed old adherent pleurisy.

A moderate amount of fat surrounded the heart. The pericardium externally appeared thickened at the apex. On opening, it contained a small amount of fluid. At the apex over an area the size of a five-shilling piece there was fibrous adherent pericarditis. The heart muscle was thinned out under the area and formed a cardiac ventricular aneurysm. The heart was enlarged, especially the left ventricle, this enlargement being due to dilatation of the cavity with thinning of the muscle. The muscle (especially the apex) was permeated with fibrous tissue, and there was some endocarditis with adherent blood clot over an area the size of a half-crown. The aortic and mitral valves appeared

efficient, but there was some simple atheroma. It did not seem possible that these valves could be the cause of the ventricular condition. There was no occlusion of the coronary arteries.

Examination of the ribs did not show any site of fracture in the bones. The

Figure illustrating traumatic aneurysm of the left ventricle, with apical myocardial fibrosis and localized endocardial thrombosis.

cartilaginous portion of the fifth, sixth, and seventh ribs over the heart apex was irregular and appeared to have been depressed slightly. There was a localized patch of pleurisy on the left parietal pleura posteriorly between the eleventh and twelfth ribs, in size about one and three-quarter inches in diameter. This patch of pleurisy was raised and reddish-yellow, and covered with granul-

ation tissue ; it was evidently the cause of the pleural effusion, and was in direct line from back to front with the pericardial adhesion. A search was made for a rib fracture in the neighbourhood of this patch of pleurisy, but it could not be found.

Death was due to cardiac failure. This cardiac failure was caused by degeneration of the heart muscle with consequent dilatation (aneurysm) and endocarditis of the left ventricle. The degeneration seemed to have spread from the pericardial adhesions. These adhesions and the localized pleurisy at the back were in direct line with the abnormality of the costal cartilages in front, and could have been the result of an impact on the chest. From the history of the case, the injuries sustained in the motor car accident were the original cause of the sequence of events that led to his death. There was, however, old-standing pleurisy on the right side, and some of the heart disease possibly antedated the accident.

COMMENTS

(1) *Clinical Aspects.*—Although this patient was obviously suffering from a grave cardiac condition throughout his illness, the true nature of the lesion was not fully appreciated, nor was a sudden death anticipated. Taken generally, the clinical picture resembled that of coronary thrombosis, except that a subacute form of " status anginosus " seemed to have persisted intermittently for several weeks. The constant tenderness over the præcordial area appeared to indicate damage to the underlying cardiac musculature.

An interesting point in the clinical history was the delay in the onset of cardiac symptoms after the accident. It appears, however, that a latent period is not an unusual finding ; for instance, Elkin (1935) cites a case of cardiac contusion following a motor-car collision, where the patient walked for a distance of 50 yards from the scene of the accident before he experienced shortness of breath. Beck (1935) also states that symptoms of cardiac injury may be delayed for hours and sometimes for days. Presumably in the present case the patient's heart was actually contused at the time of the impact ; but symptoms did not become manifest until the area of contusion, owing to extravasation of blood in the muscle, simulated a myocardial infarct.

The occurrence of auricular fibrillation following direct trauma to the chest wall has been observed by Barber (1938), White (1937), the Khans (1928), and others, and therefore it cannot be regarded as an unusual complication. The development of " tic tac " heart sounds in cardiac trauma has been shown by Bright and Beck (1935) to occur experimentally in dogs, and the same writers have observed a similar phenomenon in human beings following non-penetrating injuries of the thorax. A very striking feature in the present case was the peculiar short " whiffing " type of breathing, which may perhaps have been of some diagnostic significance though at the time it was unaccounted for. The state of anxiety and nervousness shown by the patient was seldom absent and on several occasions amounted to a veritable " angor animi."

As no electrocardiogram or X-ray photograph was taken, one can only

speculate on the possible findings. The post-mortem lesion suggests that during life an electrocardiographic tracing might have shown inversion of T in lead I, indicative of damage to the anterior and apical portion of the left ventricle, which might have been misinterpreted as pointing to a true coronary thrombosis. On the criteria given by Parkinson, Bedford, and Thompson (1938) it is unlikely that an X-ray photograph would have disclosed the presence of an aneurysm, for the heart as it lay *in situ* did not show marked bulging of the ventricular wall.

(2) *Medico-legal Aspects.*—Fortunately for most of those concerned, the case was settled out of court with substantial monetary compensation to the dependants of the deceased. There is no doubt that had the case come to trial certain difficulties might have arisen. The history of an injury in a previously healthy man would of course have carried considerable weight in a court of law, but it would have been impossible, without the post-mortem findings, to state definitely that no coronary disease had existed previously. If some coronary disease had been disclosed at the post-mortem examination it would have been rather difficult to decide how far the injury had aggravated a pre-existing disease so as to cause fatal outcome. Should the patient have survived, the question of assessing compensation might have proved even more difficult.

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ANGINA PECTORIS FOLLOWING A CRUSHING ACCIDENT

BY

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From the National Hospital for Diseases of the Heart

Received January 27, 1939.

Trauma, it is generally agreed, plays an unimportant part in the ætiology of heart disease, but occasionally, as in the preceding paper by O'Farrell (1939), an injury provides an interesting and unusual problem. In the case that follows, the absence of any electrocardiograms soon after the accident makes the evidence incomplete, but it is the best instance seen by the writer of an injury producing a picture very similar to that of a cardiac infarct.

A man of 47 years felt very fit and was able to do heavy work as a furniture-remover without any symptoms ; he had actually won a race of a hundred yards at some local sports four years before although he then weighed 14 stone. He gave no history of any previous illnesses.

One afternoon when he was unloading furniture, a fireplace surround weighing 3 cwt. slipped from the van and fell against his chest. It struck him just to the left of the mid line, inside and above the apex beat, and pushed him backwards, but a wall behind him prevented him from falling. He was imprisoned in this position for five or ten minutes, crushed between the wall and the fireplace surround. The pain was not severe ; in fact, he said that he felt nothing that could really be called pain. When he was released he felt shocked and shaky ; he sat down and rested for an hour, but still does not remember feeling any special pain. It was then an hour before work finished for the day, and he carried on for this time feeling fairly all right, but naturally not doing anything heavy.

When he had finished work and locked up, two hours after the accident, he started to walk home. After about 200 yards he had very acute pain in the middle of the sternum, which immediately made him stop until it passed off. This was the first real pain he had experienced, and it was accompanied by dyspnœa ; it recurred five or six times on his way home. He tried to do some work the next few days but could hardly do anything, and the pain got worse, i.e. more easily provoked, even on walking about slowly. After four days it seemed as bad as ever, so his doctor sent him to bed for two weeks. There had been no attack of pain that was outstanding in its length or severity, but all were very severe and he could hardly say if they were worse the first evening or during the next four days. This rest at once stopped the attacks of pain, but

they returned again when he started to get up and about. He was sent to hospital and was advised to rest for three months ; he did not have an X-ray examination, but there seems to have been nothing to suggest a fractured rib. He spent this three months quietly at home and was free from pain so long as he did not try to do much.

He was first seen by me at the National Hospital for Diseases of the Heart in October 1932, four months after the accident. He felt he was improving, but complained that on any exertion, especially on walking quickly, he had a sharp pain, which started near the left costal margin, passed up the middle of the sternum to the throat, and made him stop until relief came in two or three minutes. There was some dyspnœa with the pain, but both were less severe than at first. The cardio-vascular system cannot have been normal before his accident, as the aorta was rather widened and atheromatous and the retinal arteries were thickened ; the heart was probably a little enlarged, the maximum transverse diameter being 12·5 cm. in a chest of 24·5 cm., although its rather horizontal position made the determination of slight enlargement difficult. The only other abnormal physical signs found were a blood pressure of 150/100 mm. and an electrocardiogram showing a flat T 2 and a slightly inverted T 3.

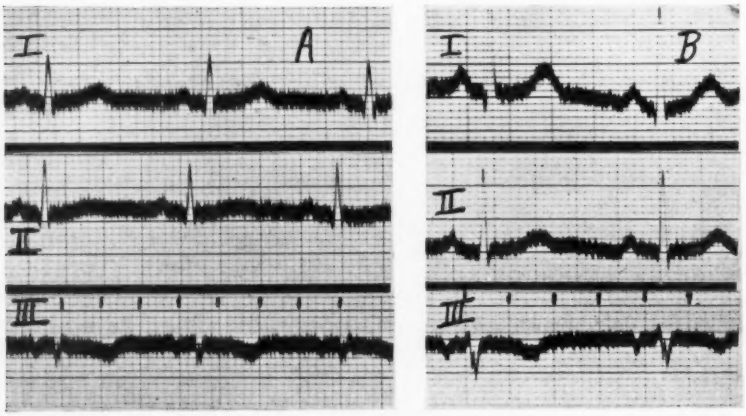

FIG. 1.—Electrocardiograms taken (A) four months and (B) five years after the accident. The improvement in the T waves, in spite of the heart having increased in size, suggests that the T waves may have been even flatter or inverted just after the accident.

After six weeks he was allowed to resume light work, as though the pain still returned if he exerted himself too much, he felt quite well when he did not do anything strenuous. The blood pressure, which had been 150/100 mm., had fallen to 130/90 mm., so probably some of the rise on the first occasion was temporary and due to excitement. He was fortunately an old and valued servant of the firm, so was given very easy work wrapping up and despatching parcels.

He was not seen again till 1937—after an interval of five years. He felt all right if he led a quiet life, but still got pain on any exertion. This started in the

epigastrium and passed up to the throat with a sensation of choking and made him stop, until it had passed off after he had rested for a few minutes. The pain was more likely to come on walking than at work ; but ever since the accident he had continued to do the same easy work, which did not involve any heavy exertion. On examination his heart had increased 2 cm. in size to 14·5 cm., but the increase in weight, from thirteen to fourteen and a half stone, might have been partly responsible. The second sound was accentuated in the aorta area, and the blood pressure was 160/100–105 mm., a little higher than in 1932. His electrocardiogram showed large upright T waves in leads I and II, with some inversion of T in lead III. It is unfortunate that no complete series of curves had been taken in the first few weeks after the accident, as they might have proved the presence or absence of an actual infarct ; but in 1932, four months after his accident, T in lead II had been almost flat and T in lead I smaller, so it seems likely that there had been earlier changes similar to those found after cardiac infarction.

At this stage the case was taken to court under the Workmen's Compensation Act and he was awarded £500 and additional payment for the time he had been off work ; he was allowed to continue the light work he had been performing for five years. The opinion expressed by the writer was that an actual thrombosis in a coronary artery or other localized damage in the heart muscle had resulted from the accident and within a short time of it ; and that his anginal pain was due to this cause. It was admitted that his cardiovascular system might not have been normal before the accident, but he had been able to do heavy work without any cardiac symptoms.

After another two years (1939) his condition has not changed greatly. The T waves in his electrocardiogram have become a little flatter, though perhaps the pain is less easily provoked. He is still doing the same easy work. There has been no further increase in the size of his heart.

Barber (1938) has reported twenty instances of heart disease that he attributes to trauma or strain, seen in the course of thirty years' practice. In two of these, both elderly men, anginal pain followed an injury; one had a fracture of the sternum where he was struck by the shaft of a cart, and the other had bruised his ribs from jumping on a moving cart. Both had to rest after the accident and the anginal pain was first noticed when they started walking three and two weeks respectively after their accidents. It is not clear how long the pain persisted. He quotes a third example reported by Fraser (1929).

Summary

A man who was leading an active life and doing heavy manual work without any cardiac symptoms, developed sternal pain within two hours of a crushing accident ; and this pain continued afterwards and was associated with some progressive increase in the size of his heart. There seems no doubt that the actual damage done to the heart at the time he was crushed was responsible for these symptoms and signs.

The commonest severe injury to the heart in crushing accidents is rupture of muscle fibres, sometimes through the whole thickness of the muscle wall; and in this case rupture of muscle fibres and extravasation of blood might account for all that was found without an actual thrombosis. But the fact that typical anginal pain, which he had never experienced before, occurred so soon and persisted for so long after the accident makes it likely that an actual thrombosis was produced while he was imprisoned, and that as soon as he started to walk after this he felt anginal pain. Alternatively, an injury or contusion of the artery at the time he was crushed might have been followed by an actual thrombosis two hours later, when he first felt severe pain. In either case, his symptoms were just those that would be expected after a cardiac infarct, and he seemed to have been in perfect health up to the time of the accident.

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A CASE OF MASSIVE HÆMOPERICARDIUM WITH RECOVERY AFTER PARACENTESIS

BY

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From the General Hospital, Colombo

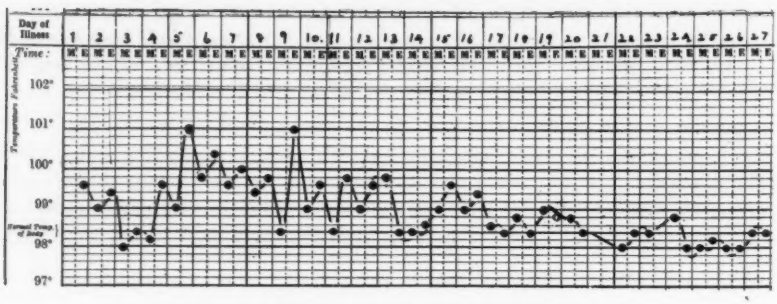
Received February 24, 1939

A young woman, aged 20 years, was admitted to the General Hospital, Colombo, under my care on June 4, 1938, with a history of having received a blow with a fist on the front of her chest the previous day. As regards her previous history, she had had pneumonia four years before and some attacks of malarial fever. She had not been in good health during the preceding six months, and attributed this to a recent pregnancy which had ended in an abortion at the fourth month.

On admission she was pale and dyspnœic. Her pulse rate was 100 and low in volume and tension. The respiration rate was 48 per minute. The lungs were resonant and the air entry was normal, with no adventitious sounds. The heart sounds were feeble and muffled. The apex beat was not palpable. Percussion of the heart revealed a large area of dullness, extending from the left axillary line to the right mid-clavicular line. A hæmopericardium was suspected. There were no injuries to the sternum or ribs. The liver and spleen were not enlarged. There was an enlarged lymphatic gland in the left axilla. This gland was excised subsequently and the pathologist reported that it was one of tuberculous adenitis with caseation. A radiogram (Fig. 1) was taken, and the radiologist reported that the appearances suggested a large pericardial effusion. The diagnosis of hæmopericardium was confirmed by Dr. H. O. Gunewardene also, who kindly saw the case in consultation.

On the following day the condition of the patient was slightly worse. There were periodical exacerbations of dyspnœa while she remained propped up in bed. An ice-bag to the chest and morphia gave her some relief. On the third day her pulse rate was 120, and dyspnœa continued. Paracentesis was decided upon and performed, and six ounces of dark sanguineous fluid was aspirated. Dr. de Saram, the pathologist, reported that the aspirated fluid was blood and that it contained no organisms and no pus cells. The patient felt slightly relieved after aspiration, and it was followed by a gradual slowing down of the pulse and respiratory rates. Her temperature, which was slightly raised at first, became rather higher for four or five days and then began to subside (Fig. 2).

FIG. 1.—Radiogram taken shortly after admission to hospital showing a large pericardial effusion and a small hydrothorax at the left base.


Day of Illness
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27
Time:
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On the advice of the Senior Physician, Dr. J. R. Blaze, nothing further was done, apart from keeping her on sedatives. The area of cardiac dullness diminished from day to day and in a week it was nearly normal (Fig. 3). At

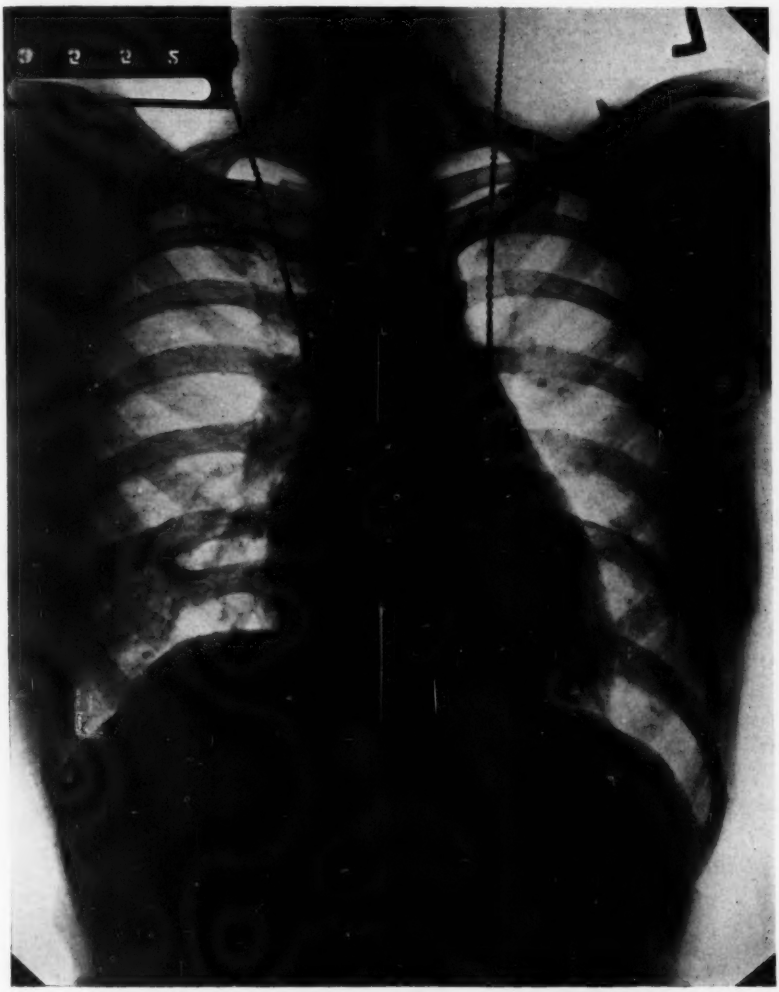

FIG. 3.—Teleradiogram (two metres) taken about a week after aspiration.

the end of a fortnight it was quite normal, and this was confirmed by a two-metre radiogram (Fig. 4).

The patient left hospital on August 17 completely cured, with a normal pulse rate and no murmurs. She was seen three months later in good health and was quite able to attend to her household duties.

FIG. 4.—Teleradiogram (two metres) taken about two weeks after aspiration.

Discussion

The case is of unusual interest because a large hæmopericardium or a large hæmorrhagic pericardial effusion had been caused by a blow which produced no external injuries.

The rapid absorption of a considerable amount of blood in the pericardial sac, determined clinically by the reduction of the extensive area of dullness almost to the normal limits of cardiac dullness within a week after the aspiration, strongly suggests that the pericardium was in a previously healthy condition. The presence of a tuberculous gland may suggest the possibility of a tubercular pericardium, but the complete return to its normal limits as shown in the second two-metre radiogram (Fig. 4) puts this possibility out of court.

The source of bleeding is a matter of conjecture. There was no evidence of hæmophilia or other blood disease.

The pathologist's report on her blood was as follows :—

Bleeding time : $2\frac{3}{4}$ minutes.
Coagulation time : 3 minutes.
White blood corpuscles : 8,800 per c.mm.
Red blood corpuscles : 3,430,000 per c.mm.
Differential count : Polymorphs, 74 per cent.
Lymphocytes, 23 per cent.
Eosinophils, 3 per cent.

Judging from the steadily progressive nature of the bleeding it was reasonable to surmise that there was bruising of the parietal and visceral layers of the pericardium. There might have been a sub-serous contusion of the musculature of the heart. The latter is, however, unlikely owing to the absence of any effects afterwards.

The successful treatment of this case by aspiration shows that operative treatment is not essential in every case of traumatic hæmopericardium, and that the prognosis is sometimes better than that suggested by Price (1937). Natural arrest of bleeding probably takes place when the intra-pericardial pressure reaches a certain point, and it is therefore probably wise to temporize with sedatives and an ice-bag to the chest in the hope of arresting hæmorrhage. If cardiac distress is nevertheless acute and progressive, operative treatment would be necessary.

Summary

1. A case of traumatic hæmopericardium without external injuries is reported.

2. Treatment by aspiration was successfully carried out.

Note by Editor. Gunewardene (1934) reported a case of a boy whose chest was crushed by a bullock cart. He returned to school and was able to play games without any pain or other symptoms. Eleven days later he com-

plained of some præcordial pain and fell down dead. There was a recent rupture of the left ventricle and it was thought to be a tear in the previously damaged muscle. Barber (1938) reported two somewhat similar cases where death took place after 4 weeks and after 6 weeks respectively.

Mayer (1936) reported seven cases seen in hospital in a period of two years. Most of them were stab wounds: in three the bleeding came from stab wounds of the left ventricle, and in three there was thought to be an anterior mediastinal hæmatoma. Five of these cases recovered and two recovered without surgical treatment, as in the case reported here.

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